The Journal of HEREDITY

Papaya Intersexes

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Looptailed Mice

Brachydactyly in Japan

Temperature Tolerances in Trout Hybrids

Yellowish-White Plumage in the Red Junglefowl

VOLUME 44 MARCH-APRIL, 1953 NUMBER 2

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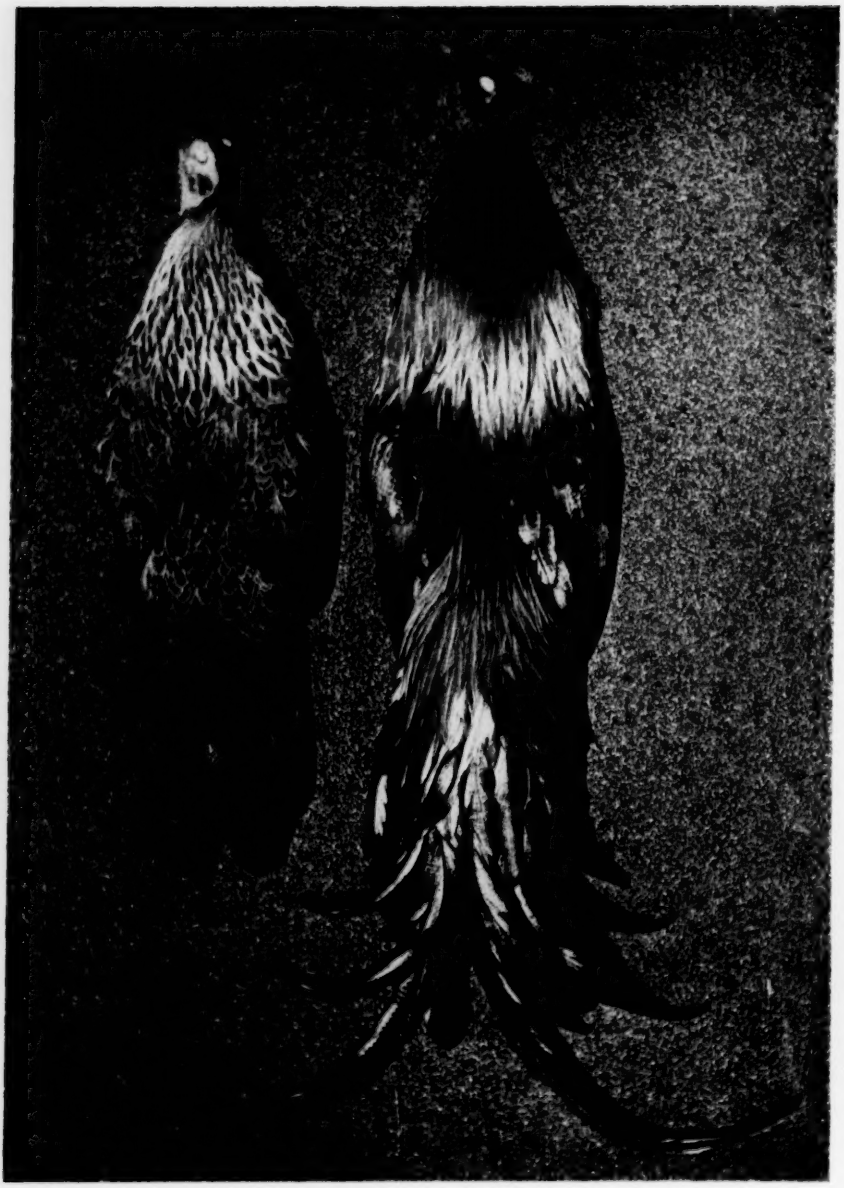


RED JUNGLEFOWL

Figure 1

Adult Red Junglefowl hen (left) and adult Red Junglefowl cock (right). These birds are typical of the Red Junglefowl (*Gallus gallus*).

A GENE FOR YELLOWISH-WHITE DOWN IN THE RED JUNGLEFOWL

G. VICTOR MOREJOHN*

IN the fall of 1946 a pair of young Red Junglefowl (*Gallus gallus*) was purchased from two independent game bird breeders. The cock was of Siamese origin and the hen of Indian origin. An approximation of the color of this species, which is widely distributed in Asia, can be seen in the domestic Brown Leghorn. Various names have been given to the complex of colors found in these fowls. The most commonly used names are: Red, Black-red, Black-breasted Red, and Brown. The first three of these are based on the colors of the cock and the last on the color of the hen.

Description

The Red Junglefowl, like the other three species of the genus *Gallus*, exhibit a high degree of sexual dichromatism. The cocks have long, lanceolate, orange-yellow feathers on the neck and rump; dark red backs; black breasts; bay-colored secondaries; blackish-brown primaries; and metallic-green tail feathers (Figure 1, right). The hens, strikingly different, are uniformly greyish-brown with blackish-brown primaries and tail feathers; light, salmon-colored breasts; and short, yellow-orange neck hackles (Figure 1, left).

The action of the gene to be described, affects the down color of both sexes and the juvenile, sub-adult, and adult plumages of the hen. Adult cocks are not affected.

The Siamese cock and the Indian hen were mated in the spring of 1948. The F_1 progeny were typically colored in the down, juvenile, sub-adult, and adult plumages. However, after three F_1 hens were back-crossed to their Siamese parent; and after two of these three F_1 hens were crossed to one of their male siblings, a small proportion of each progeny had chicks that were yellowish-white in down color (Figure 2). Yellowish-white chicks had no head stripes and only a slight indication of the brown, mid-dorsal stripe. The outer edges of the mid-dorsal stripe, which are often black, and the dorso-lateral, black stripes were variable in expression. In a few individuals the down was entirely yellowish-white with only a blackish-brown spot on the back of the head. The general appearance of the color of the down seems to be an extreme dilution of buffs and browns.

Yellowish-white and striped wild type chicks of the same age were closely observed during the growth of the juvenile, sub-adult, and adult plumages. Male and female striped wild type chicks developed similar juvenile plumage, which was like the adult, greyish-brown hen. As the sub-adult plumage developed the sexes became distinct due to the emergence on males of the black feathers on the breast and the yellowish-orange feathers on the neck and rump. The yellowish-white, female chicks acquired a juvenile plumage which was creamy-buff stippled with dark brown; and the yellowish-white male chicks acquired a juvenile plumage which was, in color, like the sub-adult plumage of the normal males. Males homozygous for the yellowish-white gene differ in this respect from the normal males in that their juvenile plumage does not resemble that of the adult female plumage. These males developed an adult plumage indistinguishable from the typical, adult Red Junglefowl cocks (Figure 3, right). Adult hens homozygous for the mutant gene, however, were atypically colored. The breast, back, and wings, except the primary and secondary remiges, were creamy buff with some dark stippling

*University of California, Davis, Calif. Grateful acknowledgment is extended to Drs. V. S. Asmundson and W. E. Howard for critically reading the manuscript; to Dr. G. L. Stebbins for helpful suggestions; and to Dr. E. M. Plocher for the gonadectomies performed.

on the feathers of the back and rump. Primaries, secondaries and retrices were black and not blackish-brown as found in the typically colored Red Junglefowl hens. Hackles of the neck instead of being yellow-orange were orange-red. These hens (Figure 3, left) resembled in color hens of the New Hampshire breed.

Genetics

Yellowish-white chicks from heterozygous parents were produced in an approximate ratio of three striped wild type (41 individuals) to one yellowish-white (13 individuals) as shown in Table I, matings 2 and 3, and the value of *P* falls between .80 and .90. There is no evidence of sex-linkage. This mutation, therefore, is autosomal, unifactorial, and recessive to the striped wild type down. When a homozygous cock and hen both of the mutant genotype were mated, all of the 28 chicks produced had the same yellowish-white down. Nineteen of the 28 chicks were raised to maturity. The hens developed the atypical, female plumage, and the cocks gradually acquired the typical, wild type plumage of normal Red Junglefowl cocks. Mating of the Siamese cock with a homozygous mutant hen produced six striped wild type chicks and six yellowish-white chicks confirming his heterozygosity for this gene. The Indian hen, however, appeared to be homozygous normal for when bred to a homozygous mutant cock 15 striped wild type chicks were produced.

Hormonal Influence

Absence of ovarian hormones following ovariotomy as shown from studies by Domm,[4] Greenwood and Blyth,[6] Quinn and Burrows,[10] and others reduces the female to the neutral poularde state which in feather structure and color is like that of the male bird. It is probable, therefore, that female plumage structure and, in some breeds, also color is governed by ovarian secretion (Taylor,[11] Hutt[7]). An experiment was devised to determine, (1) whether the action of the gene was brought about by a threshold reaction of an ovarian hormone causing the atypical plumage color in the adult female and having no effect on the adult cock, or (2) whether the action of the gene depigmented the hen and was suppressed in its action by a male hormone. Since ovariotomy is a difficult and often fatal operation which, if successful, may yield poor results due to the development of the rudimentary right gonad, cockerels were gonadectomized and given stilbestrol pellet implants.

Eight, 52 days old cockerels were used. Four of these were homozygous for the yellowish-white gene and four were normals. The experimental design is shown in Table II.

The feathers were plucked on the posterior spinal tract, the left lateral tract, and the left caudal tract on all birds so that the entire, new, developing feathers could be observed closely. At the end of four weeks new feathers had grown out sufficiently to be classified easily when compared with the plumage of normal hens and hens homozygous for the mutant gene. Gonadectomized normal cockerels and cockerels homozygous for the mutant gene developed typical wild type, cock plumage; whereas the normal and homozygous mutant cockerels that were gonadectomized and given stilbestrol pellet implants developed normal hen plumage and atypical hen plumage, respectivley, in the areas that had been plucked and at the bases of all feathers that continued to grow after the implantation. Normal cockerels and homozygous mutant cockerels given only pellet implants developed likewise.

Results of this experiment, as summarized in Table II, indicate that the mutant gene in a homozygous state requires an ovarian hormone for complete action in diluting or partially depigmenting the juvenile, sub-adult, and adult plumages of females. The gene shows no effect whatever on the plumage of adult cocks. Hens of some Oriental Black-red Shamo games and the Black-breasted Red Cubalaya show this same striking sexual dichromatism. Whether the chicks of these breeds are yellowish-white or not is not known by the writer.

TABLE I. Results of matings between a Siamese Red Junglefowl ♂, and Indian Red Junglefowl ♀, and their descendents

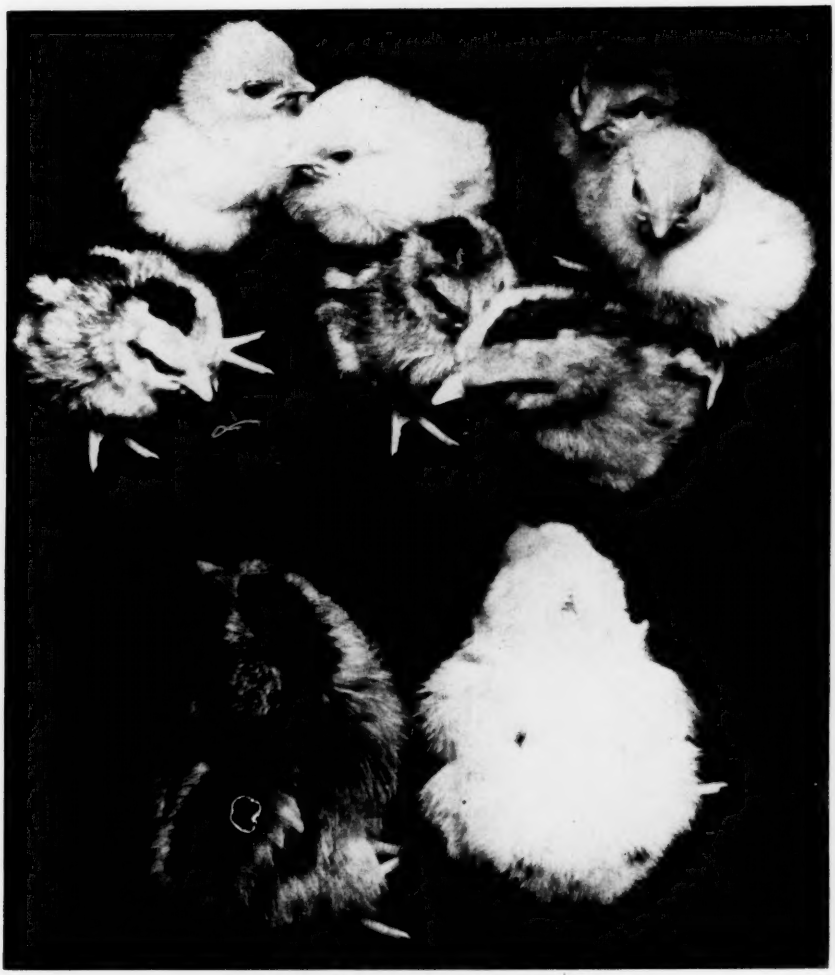
Matings	Progeny: Wild type down ♀♀	♂♂	Total	Yellowish-white down ♀♀	♂♂	Total
1. Indian ♀ × Siamese ♂	4	3	7			0
2. F_1 ♀♀ × Siamese ♂†	7	8	15	1	1	2
3. F_1 ♀♀ × F_1 ♂†	8	8	26*	2	3	11*
4. Mutant ♀♀ × Siamese ♂	..	..	6*	..	..	6*
5. Indian ♀ × mutant ♂	..	..	15*	..	..	0
6. Mutant ♀♀ × mutant ♂	..	..	0	10	9	28*

*The totals are greater than the sum of the male and female totals because the sex of some or all of the progeny was not determined.

†Cock crossed with hens individually. All were heterozygous for the mutant gene.

TABLE II. The effect of castration and stilbestrol pellet implantation on the plumage of four normal and four mutant Red Junglefowl cockerels

Type of cockerel	Treatment	Type of new feathers
Normal (control)	None	Normal cock
Normal	Stilbestrol	Normal hen
Normal	Castration	Normal cock
Normal	Castration & stilbestrol	Normal hen
Mutant (control)	None	Normal cock
Mutant	Stilbestrol	Mutant hen
Mutant	Castration	Normal cock
Mutant	Castration & stilbestrol	Mutant hen



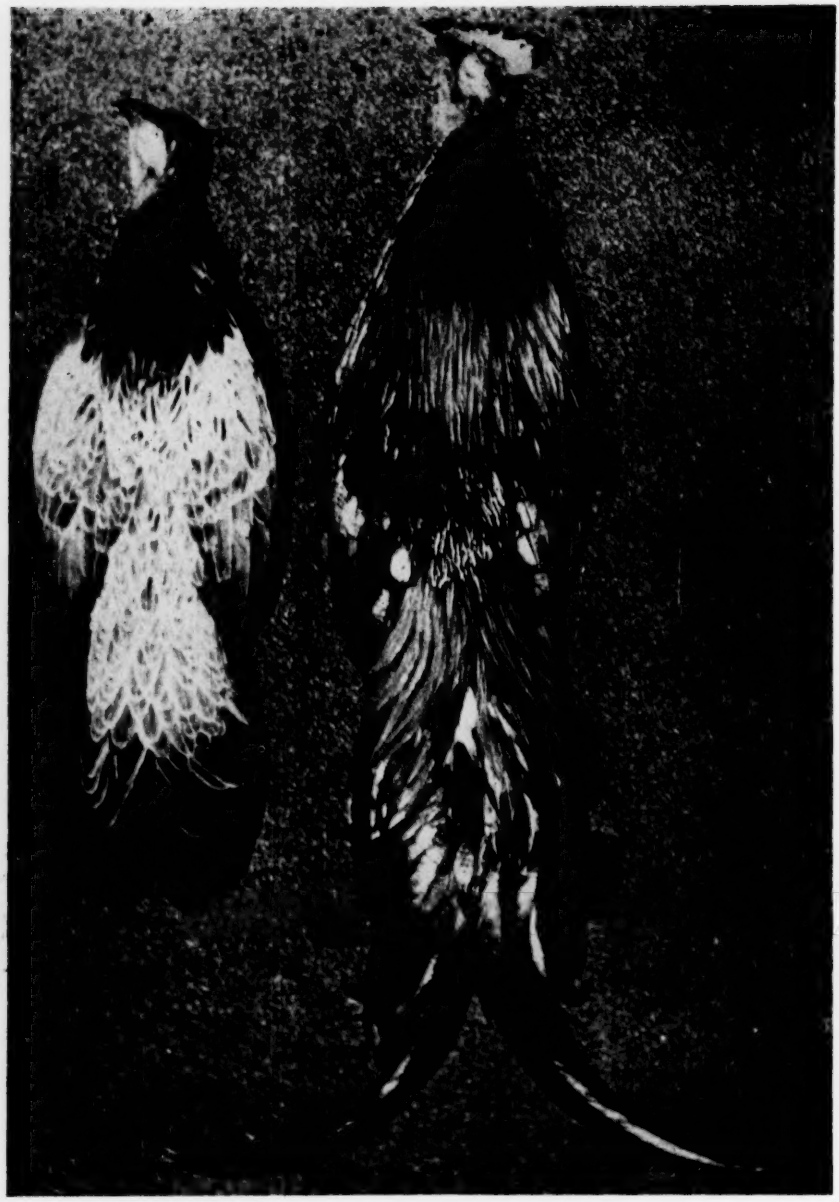
DAY-OLD RED JUNGLEFOWL CHICKS
Figure 2

Normal, striped wild type chicks and yellowish-white chicks (above). The mid-dorsal and dorso-lateral stripes are clearly visible in the normal chick (left, bottom), and the expression of these stripes on these yellowish-white chicks (right, bottom) was variable. The white chicks proved to have a very low survival under natural conditions.

Discussion

Bateson and Punnett[1,2] found a similar type of yellowish-white down in the F_2 progeny of the Brown Leghorn crossed with a recessive white fowl. Brown Leghorns have the same striped wild type down pattern as found in the Red Junglefowl chicks. Recessive white fowls carry the recessive gene *c* for prevention of color development, and do not show the hidden plumage types and down patterns which they may carry. These hidden characters can only be revealed by proper breeding tests. In the cross made by Bateson and Punnett, the F_1 chicks were all striped wild types; but the ratio among the chicks produced in the F_2 generation was nine striped wild type chicks : three unstriped pale brown chicks : four unstriped white chicks.

The unstriped, pale brown down is inferred by Hutt[7] (p. 218) as probably caused by a recessive allele *st* of the striped wild type



RED JUNGLEFOWL HOMOZYGOUS FOR YELLOWISH-WHITE GENE

Figure 3

Mutant Red Junglefowl hen (left), which differs in plumage color from the normal hen, and mutant Red Junglefowl cock (right), which is indistinguishable from normal cocks.

down carried by the recessive white fowl, and for the dominant allele the symbol *St* (stripe) was proposed. The unstriped, pale brown chicks as adults could not be distinguished from the Brown Leghorns which had been striped wild types as chicks. The unstriped white chicks presumably were the extracted recessive whites, since no mention was made of their adult phenotypes. The gene mutation reported for the Red Junglefowl in this paper clearly has no relation with the recessive white condition and must be another recessive allele of the striped wild-type down pattern. The chicks of the Dark Cornish breed of domestic fowl have greyish-white down with a striped pattern on their backs which is similar to the striped wild type discussed, but many of the chicks show no head stripe at all. Many other domestic breeds of fowl exhibit some form of striping of the back and/or head stripes in the down. Kimball[8] has studied the down patterns and colors of the down of several domestic breeds. He has found some of these to be caused by separate genes.

The symbols proposed by Hutt[7] for the striped and unstriped patterns of the down, based on Bateson and Punnett's data, may be confusing when the many other down patterns that show some form of striping are analyzed. The down pattern of the chicks of the Red Junglefowl (if the latter is to be considered the original wild ancestor of domestic fowls), therefore, should have a more appropriate name than "stripe" to designate the head stripe and back stripes of the wild chicks. Since Kimball[9] has assigned the symbol e^+ to the Red Junglefowl wild type adult plumage and its domestic derivatives, and has found e^+ to be allelic to *E* (extended black) and *e* (Columbian restriction) then the mutation described in this paper could possibly be another member of this triple allelic series. The gene *St* proposed by Hutt for the striped pattern in the down, therefore, may be the same gene, e^+, described by Kimball for the black-breasted red plumage color of the adults. If conformity in designation of symbols is to be followed as suggested by Dunn, Gruneberg and Snell,[5] then the symbol e^+ or St^+ (for *wild type* striping) should stand as the poultry geneticist's "wild type." No symbol is assigned to the yellowish-white down described, for evidence of allelism with *E* (extended black) is not yet available. Linkage tests and tests for multiple alleles are in progress.

The existence of this gene in natural populations is not known. Probably it is restricted to a small percentage of heterozygous individuals; and, since dominance is complete, natural selection for these heterozygous birds would not be possible. Examples of hidden, recessive mutations carried by heterozygous individuals in natural populations have been discussed by Dobzhansky[3] (pp. 63-65). Birds homozygous for the mutant gene would have an extremely low survival value due to their yellowish-white down as chicks. A simple experiment relative to this point was carried out. A typical wild type hen was turned loose with six striped, wild type chicks and six yellowish-white chicks. The area afforded cover and native grasses, but food and water were supplied. By the end of the first day two yellowish-white chicks had been lost due to the depredations of blue jays. When the hen and chicks were observed the third day only the six striped wild type chicks remained. Previous to the loss of the yellowish-white chicks the hen was observed several times giving the alarm call when danger threatened. At the sound of the call all the chicks scattered. Some of the striped wild type chicks could be differentiated at times among the dead leaves and grasses, but the yellowish-white chicks invariably stood out as clearly as white balls of cotton.

Summary

A typically colored Red Junglefowl cock of Siamese origin was crossed with a typically colored Red Junglefowl hen of Indian origin. The progeny resulting from the cross were typical Red Junglefowl color, but when some of these birds were crossed *inter se* chicks were produced in a ratio of three with wild type to one with yellowish-white down. When three hens of this F_1 progeny were backcrossed to their Siamese parent, chicks were also produced in a ratio of three wild type to one yellowish-white. This indicated that the Siamese cock was heterozygous for this gene, and that the F_1 hens crossed back to him were also heterozygous. Birds that had had yellowish-white down as chicks were mated and produced all yellowish-white chicks. Cocks of these yellowish-white chicks had typically colored adult wild type plumage, while the hens had atypical plumage similar to that found in the New Hampshire breed.

To determine the hormonal influence of the gonads, two normal and two yellowish-white downed cockerels were gonadectomized, and one of each was given a 15 mgm. diethystilbestrol pellet implant. Intact males of each type were also given similar diethlystilbestrol pellet implants, and one of each type was used as a control. All birds had three different feather tracts plucked. The implanted males developed plumage typical of hens of the corresponding genotype while the regenerated plumage of the castrated and intact males not implanted was typical of the wild male. The color of the plumage of the female, therefore, is determined by interaction of the gene and ovarian hormones.

Probable relations of this gene to other genes known to affect the wild type (black-red) plumage pattern in the fowl are discussed.

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(*Received September 2, 1952*)

◆

Every Science is Party Science

The Marxist-Leninist principles governing Soviet teaching methods are embodied in the "Soviet science of pedagogy," which, like other Soviet "sciences," serves political ends. "We must not forget for a moment," wrote the official Soviet pedagogic journal *Sovietskaia Pedagogika* (Moscow) in its first issue (fall 1946), "that every science is Party science." Nor are Satellite educators permitted to forget. Soviet pedagogy is faithfully followed by the captive teachers who play the role of "Socialist builders." *Rude Pravo* (Prague), August 18, 1952 wrote: "In order to bring up and educate our new Socialist generations ... the teachers . . . must bring them up on the basis of the one and only ideology and science, by which we are directed in all tasks of construction of our new life, on the basis of Marxism-Leninism. . ."

Zeri i Popullit (Tirana), September 2, 1952, in an editorial on the "New School Year," apprised Albanian teachers of their duty to follow Soviet experiences:

". . . It is their duty to assimilate very well and put into action, without incorrect interpretation, the experience and excellent teaching methods of the Soviet schools . . . (and) . . . the learning and teaching of Marxism-Leninism. The education of the teachers in this matter is absolutely imperative, because only thus can a teacher have the key of wisdom in his hands. . . ."

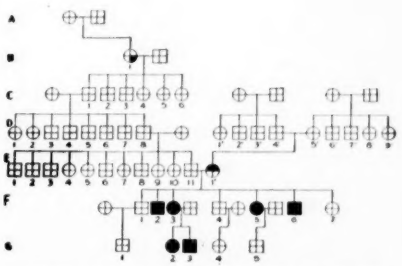
News from behind the Iron Curtain March 1953.

CLUBFOOT AND CONGENITAL HAND ANOMALIES

C. J. CAVANAUGH*

CONGENITAL anomalies of the hands and feet have received considerable attention in the literature as evidenced by the great number of papers and variety of journals in which articles have appeared. Barsky[1] points to the earliest written reference to such anomalies as about 1000 B.C. and recorded in the Bible in reference to the Giant of Gath who had six fingers and toes. A very extensive bibliography of over 400 publications will be found in the work of Birch-Jensen[3] who published a monograph dealing with the genetic aspects of congenital anomalies of the upper extremities. Stewart[7] includes a lengthy bibliography in his paper concerning the incidence, cause and treatment of clubfoot.

The etiology and occurrence of clubfoot have been considered by a number of writers and various opinions have been expressed. Fetscher[4] found from his study of 32 pedigrees that only six cases showed direct transmission from parent to offspring. He estimated the frequency of clubfoot at one in 1000-1600. Stewart[7] states that clubfoot appears to be hereditary in some ethnic groups with a greater incidence among Hawaiians than among other elements in the population in Hawaii. He finds a high incidence of one in 1,000 with a male predominance of two to one. Wright[8] calls attention to certain factors of the environment as modifiers of the developmental pattern. He considers that unfavorable prenatal conditions permit several genes to produce their effects while they would not be able to do so under more favorable conditions of development. Murphy[6] concludes, from his study of over 500 families with defective children in the city of Philadelphia, that gross human congenital malformations arise solely from influences which affect the germ cells prior to fertilization. He found in families already possessing a malformed child, that the birthrate of subsequent defective offspring was approximately 24 times greater than in the general population. Stewart[7] concludes that clubfoot is due to abnormal differentiation of the individual tendons at or near their attachments in the foot during the period between the fifth to eighth week of gestation which results in an uninhibited positive deforming force. He believes the transmission of the tendency for the abnormality has a genetic basis. Bektol and Mossman[2] describe two cases of clubfoot in which their histological findings indicated that normally developing skeletal structures



PEDIGREE OF CONGENITAL CLUBFOOT AND HAND ANOMALIES

Figure 4

Solid symbols represent the bilateral clubfoot and bilateral hand anomaly. Solid lower right quadrant represents unilateral clubfoot only. Solid upper half of symbol represents bilateral hand anomaly only.

*Department of Biology, Louisiana College, Pineville, La. This project was largely financed by funds from the Carnegie Foundation for the advancement of teaching. The author is indebted to Dr. Gene D. Caldwell and the Shriners Hospital for Crippled Children of Shreveport, Louisiana, for the pictures of Figure 5 and for helpful suggestions during this investigation.

THE CONGENITAL CLUBFOOT ANOMALY

Figure 5

At the left is shown individual F-3 and at the right individual F-6 exhibiting the clubfoot condition. These two cases are representative of the feet of all affected individuals in the pedigree.

had become displaced due to abnormal skeletal muscle development. The muscle abnormality consisted of degenerating muscle fibers which caused the muscle to fail to keep pace with skeletal growth. Gates[5] mentions the following suggestions and conclusions drawn by these writers: Isigkeit's suggestion that the gene for clubfoot is recessive sex-linked; Debrunner's case of direct transmission from mother to daughter and another from father to son with frequent skipping of generations; Debrunner's conclusion that congenital clubfoot is a monohybrid recessive that probably requires a "realization-factor" for its expression; Scherb's conclusion that several genes produce clubfoot. Gates considers clubfoot as generally recessive or in some families a dominant of very low penetrance.

The purpose of this paper is to present a pedigree believed to be sufficiently significant that it should be recorded in order that it may be used in later statistical reports. This pedigree is considered of value because it shows the apparent sudden appearance of unilateral clubfoot in one generation and a skip of three generations before a reappearance. This reappearance came when the clubfoot lineage was introduced into a lineage where it was combined with a hand anomaly. From this point on the condition has appeared in both of the succeeding generations with a ratio approximating one-half normal to one-half defective and with equal distribution between males and females.

As Figure 4 shows, the pedigree commenced with unilateral clubfoot of the equino varus type in a female. This was the first and only known appearance of such an anomaly in this family and there were no known cases in the next three generations as determined by careful questioning of members of the families, doctors, nurses, demonstration agents, shoe clerks, ministers, and other members of the communities in which they lived. However, when a third generation male married an unrelated female who possessed a hand anomaly, (see Figure 6), bilateral clubfoot (equino varus in one foot and calcaneovalgus in the other) combined with the hand anomaly appeared in four out of seven children with equal distribution between males and females. The same anomaly of hands and feet appeared in the next generation as indicated. Individuals F-3, G-2, and G-3 had equino varus of the left foot and calcaneovalgus of the right while F-2, F-5 and F-6 had equino varus of the right foot and calcaneovalgus of the left. One of these, F-2, died in infancy. All of the others are living and have had corrective surgery.

Stewart[7] stresses the need for more anatomical studies of clubfoot. Gates[5] calls attention to the fact that a series of conditions of very different etiology and anatomical structure are included under the term clubfoot. Since a thorough anatomical description of the clubfoot and hand anomaly of this pedigree is impossible Figure 5 of individuals F-3 and F-6 respectively are included. These two are representative of the feet of all affected individuals in this pedigree. From a comparison of these photographs with the descriptions and figures given by Bektol and Mossman[2] and Stewart[7] and from their history of reoccurrence after early correction it might be concluded that the clubfoot here resembles the condition described as being due to normally developing skeletal structures becoming displaced by abnormal muscular forces, either produced by faulty skeletal muscular development as described by Bektol and Mossman or by abnormal differentiation of the indi-

THE CONGENITAL HAND ANOMALY

Figure 6

A and *B*—front and rear view of the hands of individual E-1′. *C* and *D*—front and rear view of the hands of individual G-3. The hands of both persons are in a state of maximum phalangeal extension.

vidual tendons at or near their point of attachment as suggested by Stewart.

The anomaly of the hands is somewhat different from any the author has found described. It consists of a marked degree of flexion of the phalanges at the metacarpal-phalangeal articulation with some abduction of the phalanges and an inability to accomplish their extension. Figure 6 *A* and *B* shows the front and rear views of the hands of individual E-1′ in a state of maximum phalangeal extension. The anomaly appeared first in this woman and was her only abnormal development. Figure 6 *C* and *D* shows similar views of the hands of individual G-3 who is the granddaughter of E-1′. The hands of all affected individuals more nearly resemble Figue 6*A* and *B*. All individuals demonstrate a remarkable use of their hands, but display a reduction of about one-fourth in the total anteroposterior and abduction to excursions of the wrist as well as a marked reduction in ability to supinate the hands. This anomaly appears to be due to a displacement of normal skeletal structure by abnormal muscular forces.

A complete check of all generations of offspring for all individuals from families D,E and F and for most of Family C has been made. Only three other cases of deficiencies of hands or feet have appeared. In a second generation from D-9′ one daughter had unilateral camptodactyly of the right little finger, but this did not reappear in any of her three children (two females and one male) nor in her three grandchildren (all females). The only son of D-2 had short thumbs due to the terminal phalanx being very short, somewhat spatulated, and with a broad short nail. He was unable to flex his toes beyond a normal standing position and the tendon of the extensor digitorum longus of the fourth toe on the left

foot maintained that digit in a state of hyperextension. This individual also had a condition of mental deficiency without psychosis and developed a paralyzed right eyelid late in his teens. In the first generation from E-4 one son had a severe condition of pes planus which had to be corrected by surgery before he could walk properly.

It would be unwise to attempt to draw significant conclusions from this single pedigree. However, it does add further evidence to the now generally accepted view of the genetic basis for clubfoot.

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LETHAL TEMPERATURE EXPERIMENTS WITH SPECKLED TROUT × LAKE TROUT HYBRIDS

F. E. J. Fry and M. B. Gibson*

In December 1950, approximately 1,000 eyed eggs of a cross between male speckled trout, *Salvelinus fontinalis*, and female lake trout, *S. namaycush*, were received at the Laboratory for Experimental Limnology, Southern Research Station, Maple, Ontario, through the kindness of the Canadian Wildlife Service. The cross was made by Mr. J. E. Stenton, the warden of Lake Minnewanka District, Banff National Park, Alberta.

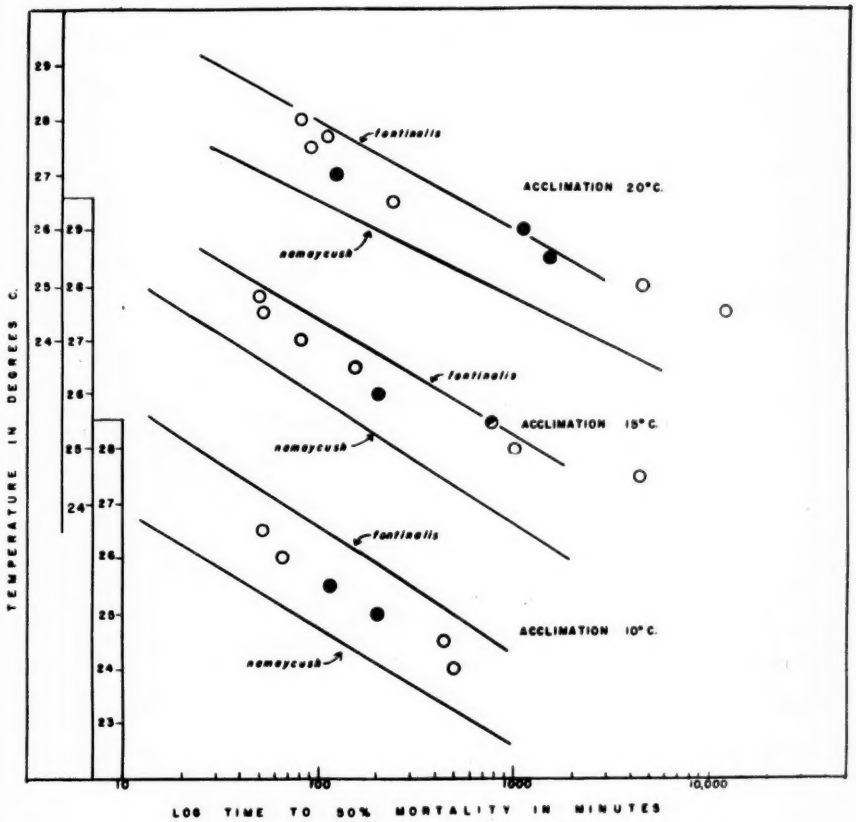
In previous years workers in our laboratory had conducted lethal temperature tests with the speckled trout[1] and with the lake trout.[2] The hybrids have been tested by the same methods as had been employed for the parent stocks. Briefly, this method is to maintain the fish for a certain length of time at a constant temperature which is referred to as the acclimation temperature. Then, samples of the acclimated fish are placed directly into baths held at the test temperatures, and the time to death of each fish is noted. In the present experiments either five or ten fish were used in the samples.

Acclimations of the hybrids to 10°, 15°, and 20°C. were begun in June, 1951, and the lethal tests were started in August and continued during the fall and winter.

The results of the tests have been plotted on semi-log paper, each point representing the median time to death of a sample of fish at a particular temperature. In the points plotted all the fish in each sample died before the experiment was discontinued. The average standard error of the mean time to death was 7.4 percent for the 20°C., 6.7 percent for the 15°C., and 6.3 percent for the 10°C. acclimated hybrid fish. A composite graph is presented which shows what happened at the three different acclimation levels. The points plotted represent the hybrid tests, black circles show samples of 10 fish, open circles those of 5 fish. The two lines drawn at each level are lines fitted by eye to indicate the slopes of similar points obtained previously for the two parent species.

Two tests were made in addition to the points shown on the graph. Fish acclimated to 10°C. were tested at 23.5°C., and in this test the fish all lived and were active for more than 4,000 minutes when the experiment was discontinued. The second test in which mortality was not complete was performed on 20°C. acclimated fish at 24.0°C. Only one out of five fish died at over 26,000 minutes. From these data together with the data in Figure 7, it is believed that the incipient upper lethal temperature[1] of these hybrids is between 23.5°C. and 24.0°C. at 10°C. acclimation and

*University of Toronto and Ontario Department of Lands and Forests, respectively. Grateful acknowledgment is made to Mr. Wm. Sanderson of the Limnology Laboratory staff who looked after the care and feeding of the hybrids and also the acclimations and overnight watching when lethal tests were in progress.



RESULTS OF TEMPERATURE EXPERIMENTS

Figure 7

Thermal resistance of the hybrids in comparison with that of the parents. Each point represents the logarithmic mean time per sample.

between 24.0°C. and 24.5°C. at 20°C. acclimation.

Both parents are hardy at 0°C. when acclimated to 20°C. A sample of the hybrids of the same acclimation was tested at 0°C. and survived for two days. The experiment was then discontinued as the fish showed only a slowing down of movement but no other signs of distress. It was concluded that the hybrids are hardy at 0°C. also.

A rather interesting trend with rising acclimations appears in these experiments. At the lowest acclimation, 10°C., the position of the line for hybrid trout is approximately mid way between the lines for the two parents. This is a circumstance that has often been observed in other comparisons of hybrids with parents with respect to form, structure, and color. At 15°C. acclimation the hybrids approach the median resistance times of the *fontinalis* parent much more closely than they do the *namaycush* parent, and at 20°C. acclimation this trend appears to be even more pronounced.

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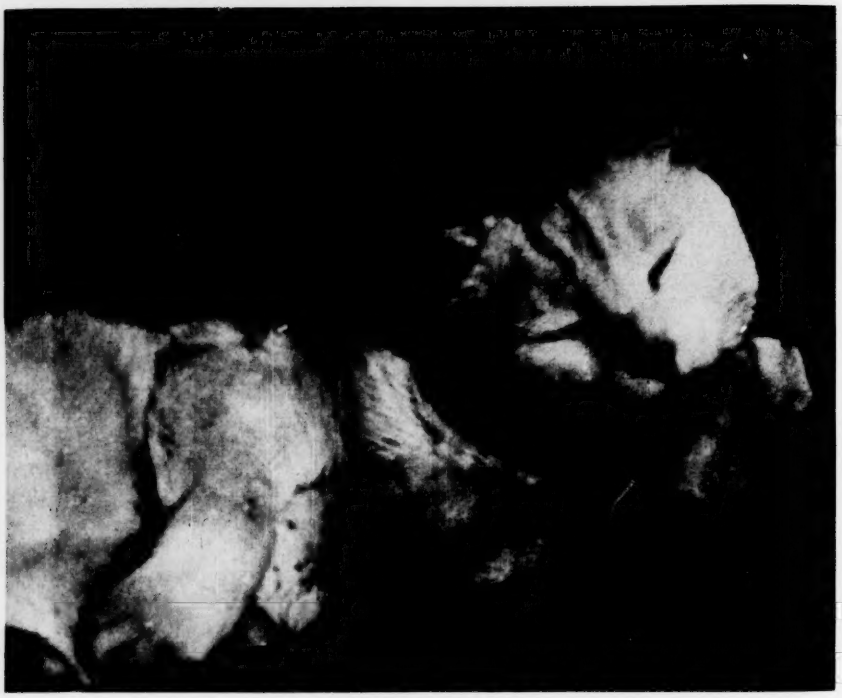
"Jew's-Harp" Marking in the Forehead of a White Cat



WHITE MONKEY

Figure 8

The typical "Jew's-Harp" marking can be seen in the forehead.

IT is well known that all cats of the tabby pattern carry the "jew's-harp" marking on the forehead. This marking appears whether the body bears the swirls of the blotched tabby pattern or the vertical bars of the lined or striped pattern.

The occurrence of the "jew's-harp" marking in the forehead of a pure white cat of mine suggests the possibility that the gene may be pleiotropic in its influence, controlling the structure of the fur's pattern as well as its coloring.

This cat of ours, the White Monkey, a blue-eyed white domestic short-hair, does not possess a single colored hair. Yet on his forehead the traditional tabby marking is plainly stamped. In his case, it is a question of furrows.

Unfortunately, Monkey can contribute no genetic evidence as regards his forehead marking. We got him as a kitten of three months without having seen either of his parents. And he can never have descendants. Either because of some congenital defect or as a result of a severe attack of gastro-enteritis from which he nearly died shortly after he came to us, he has never matured sexually. He now is ten years old.

Though not a true albino because of his blue eyes, he shows a suggestion of albinism in that his eyes glow in semi-darkness with a brilliant ruby light rather than the usual topaz, turquoise or emerald of pigmented cats. Also, viewed from certain angles in deep shadow, his eyes appear pink.

The tabby stamp on Monkey's forehead, however, though colorless, is so distinct in detail that it has led me to examine carefully the forehead of Some Pumpkins, a red and white striped tabby of our household. And I find that with Pumpkins the color follows the surface contour, the deep red of the "jew's-harp" marking being set in the furrows of the fur and contrasting with the lighter, more yellowish coloring that tips the ridges.

HOPE SATTERTHWAITE JEX
New York City

DEVELOPMENT OF MICE HOMOZYGOUS FOR THE GENE FOR LOOPTAIL

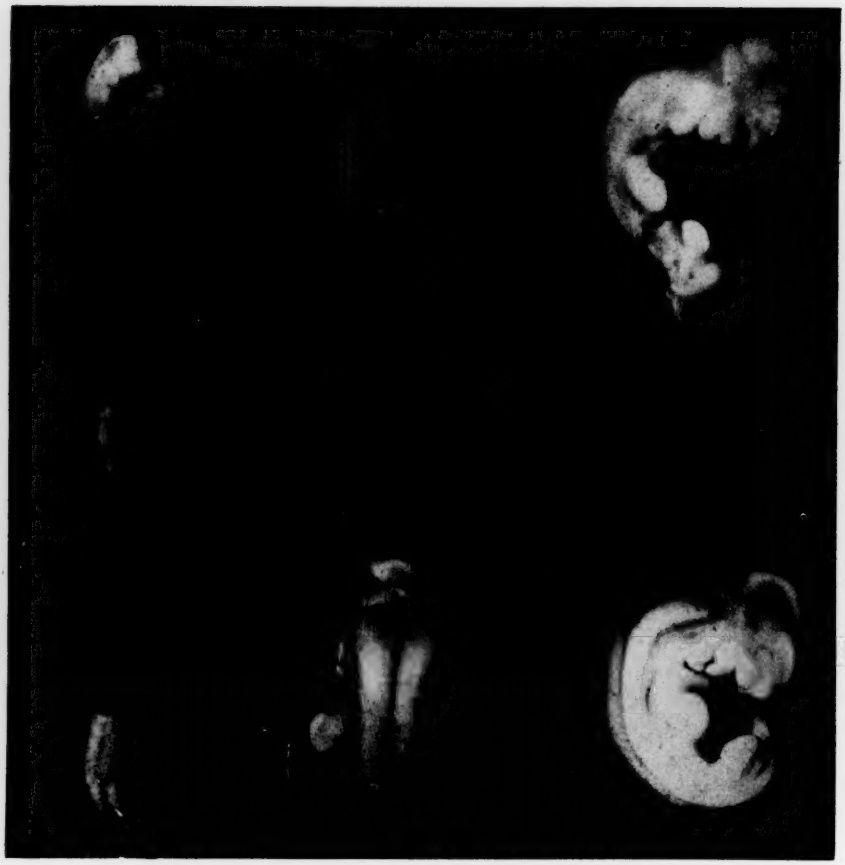
K. F. Stein and Iris Abrams Rudin*

THE mice used in this investigation were descendants of individuals of Strong's A strain[13] in which the gene for looptail occurred spontaneously in the 84th generation of brother-sister inbreeding. Strong and Hollander[14] reported the appearance of this character and also that the gene was variable in its expression, apparently dominant with incomplete penetrance, and with craniorachischisis resulting from a double dose. Homozygotes are alive in utero but are apparently eaten by the mother at birth. On these points our results are in agreement with theirs: 21 (approximately 23 percent) of the 92 embryos which could be classified for this investigation from matings of looptail × looptail, had open neural plates or folds and were considered homozygotes, the remaining 77 percent had normally closed nervous systems (Figures 9 and 10). As regards the looping effect on the tail of a single dose of the gene (Figure 11), 71.0 percent of the expected number of heterozygotes could be recognized as such in our litters compared with 63.3 percent reported from the same type of cross by Strong and Hollander. In the present investigation no attempt was made to determine the prevalence of the behavior trait "wobbly" described by Strong and Hollander and found by them in all the mice with looped tails and in some straight-tailed animals which produced looptailed offspring when outcrossed to normal mice. They considered this behavior trait another dominant manifestation of the looptail gene, which would thus appear to have greater penetrance in the head than in the tail region.

As demonstrated by Strong and Hollander[14] the homozygous dominant embryos are characterized by open neural folds in the region of the brain and may or may not have open spinal cords as well (spina bifida). However no attempt was made by these investigators to study the development of the anomaly. Consequently the present study was made on a series of timed embryos obtained from matings of heterozygotes (looptail × looptail). The genetic background was not uniform; a variety of strains were used to furnish the individual looptailed parents. Age of the embryos was determined from the day of appearance of the vaginal plug and/or by the use of Grüneberg's criteria.[6] Litters ranging in age from 8 to 18 days were examined. So far it has been impossible to distinguish the homozygous affected embryos before the middle of the 9th day (Figure 9), that is before closure of the nervous system has occurred. Closure of the neural folds begins, as in the rat,[9] at the level of the second and third somites and proceeds posteriorly. Slightly later the forebrain closes, except for the anterior neuropore; the mid and hindbrain areas are the last to close. Consequently individuals with open spinal cords as well as open mid and hind brains can be detected slightly earlier than those in which the spinal cord closes normally and only the brain area is affected. Though several litters of embryos younger than nine days were examined and sectioned no indication of which ones might have developed the abnormal brains was observed. Possibly histochemical tests may later reveal a difference.

In view of the fact that the notochord, or organizer tissue, is frequently involved when homozygous genes for tail anomalies are responsible for drastic effects on the nervous system, a careful comparison was made between sections of embryos homozygous for

*Mount Holyoke College, South Hadley, Massachusetts. The authors are indebted to Dr. L. C. Strong for furnishing mice with the factor for looptail.



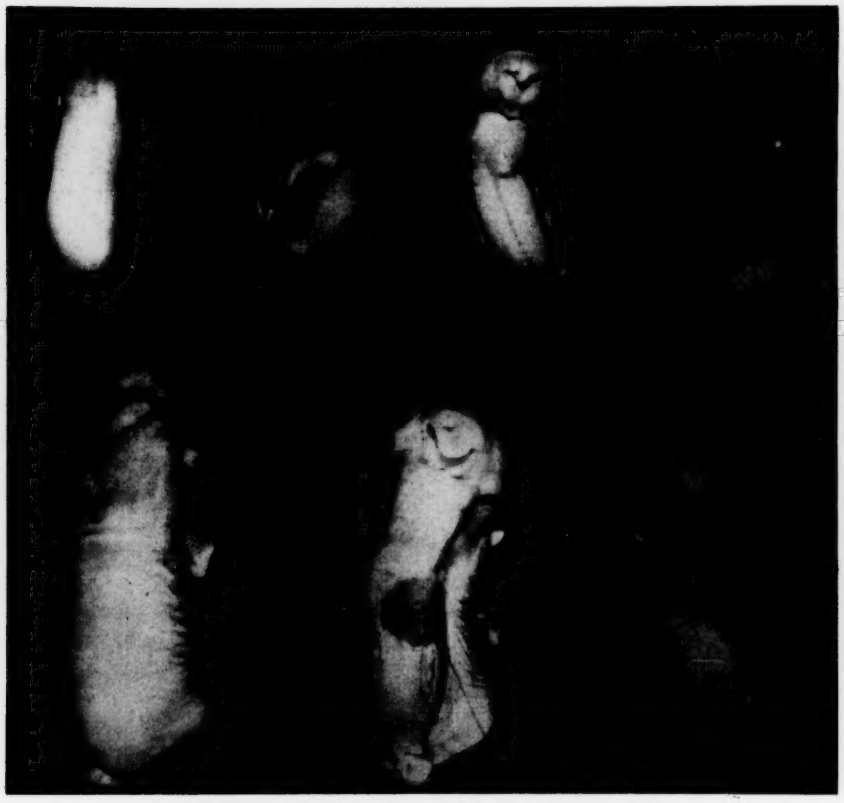
DEFECTIVE EMBRYOS AND NORMAL LITTERMATES

Figure 9

A—normal, *B* and *C*—homozygous looptail embryos nine days of age, (×1). *D* and *E*—normal, *F* and *G*—defective embryos 11 days old, (×6).

looptail and those of normal littermates at 9, 10, 11, 15, and 17-18 days. The embryos were fixed in Bouin's, Helly's or Zenker's fluid, cut at 10 to 20 micra depending upon age, and stained in hematoxylin. No difference was observed between the two types of embryos in the appearance, development and transformation of the notochord during this period (Figure 12).

The histological differentiation of the nervous tissue in abnormal embryos appeared to be normal; gray and white matter were formed in approximately similar proportions (Figures 12 and 13). The morphology however was quite different. This was obvious externally in the open portions of brain and cord of the affected mice and sections showed that even in the forebrain and in that portion of the midbrain which was closed there were abnormal space relationships (Figure 16), at least from the age of 10 days. The lumen of the forebrain was collapsed, as if by pressure from the outside or more likely from the lack of pressure normally produced from the presence of fluid within the brain cavity. The choroid plexae were present and apparently normal in differentiation; tissue of the mesencephalon protruded making up a median dorsal extension frequently seen in these embryos, or part of a large flap-like extension of neural



EMBRYOS HOMOZYGOUS FOR LOOPTAIL GENE

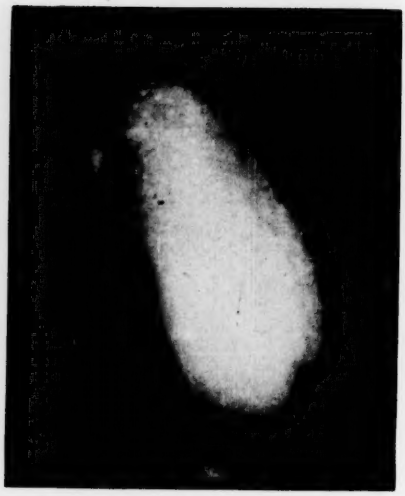
Figure 10

A and *B*—normal, *C* and *D*—defective embryos, 15 days old. *E*—defective 17½ days embryo with normal spine and open brain. *F* and *G*—defectives of the same age with excess "flaps" of neural tissue and open neural plates "punctured" by vertebral arches, (×4).

tissue which sometimes overhung the face (Figure 10). An excess of nervous tissue appeared to be present, especially in the open regions of the mid and hindbrain (Figure 16). For this reason counts were made of the number of mitotic figures in the nervous tissue between the point of emergence of the optic stalk and the level of the beginning of the auditory anlage (Table I), and also in a given number of sections at a lower level of the cord (Table II). At the cord levels no difference in the amount of proliferation was observed but there appeared to be more mitotic figures in the brain region of the abnormal embryos than were found in their normal littermates. The number of mitoses was obtained by counting figures in every 2nd, 9th, or 12th section, as indicated in Table I, and multiplying by the number of sections in the interval, e.g. by 9 if every 9th section was counted. The authors realize that the thickness of the sections and the multiplicity of divisions in the nervous tissue make for inaccuracies in the absolute numbers of figures reported, but the conclusion that there is greater proliferative activity in the mid and hind brain in the abnormal embryos, relative to that in the normal, seems justified.

Other Abnormalities

Other abnormalities noted were essentially those to be expected as sequelae of the open nervous system. In the cord region the vertebral arches could not form normally around the open flat plate of nervous tissue. That their normal dorsad growth is not completely



HETEROZYGOUS LOOPTAIL

Figure 11

Embryo 15 days old from same litter as those shown in **Figure 10 (×4).**

dependent upon the presence of a normal spinal cord is indicated by their puncture of the flattened folds in an apparent attempt to grow in the normal direction (Figures 10, 12 and 15). Other parts of the nervous system, such as cranial and spinal ganglia and nerves (Figures 12 and 13), formed normally. Pituitary and thyroid, adrenals, gonads and kidneys, liver and pancreas differentiated similarly in both types of embryos. In later stages of development the affected individuals tended to be smaller than their normal litter mates, possibly due to some interference with circulation. Hemorrhages had occurred into the amniotic cavity in some instances. Since Strong and Hollander[14] reported wobbly head behavior as one aspect of the gene action in hybrids it was considered of interest to compare normal and homozygous abnormal embryos for development of the ear. Wax reconstructions (magnification 100×) were made of the auditory anlage at 15 days of embryonic development. The various elements, utriculus, sacculus, semicircular canals, cochlea and endolymphatic duct were present and appeared superficially similar in the two embryos (Figure 14). Detailed histological study has not been made. Morphologically the entire anlage in the abnormal embryo seemed to be somewhat flattened. This may perhaps result from the lack of mechanical stimuli which would normally occur in the presence of a closing and closed hindbrain.

Discussion

The gene concerned in this investigation was observed by Strong[13] in an inbred but untreated strain in 1947, its effects were described by Strong and Hollander[14] in 1949 and compared by Grüneberg[7] with other anomalies of tail and nervous system in the mouse. It causes a loop or a twist in the tail of a certain proportion of embryos hybrid for it and a wobbly behavior of the head in a somewhat larger proportion. Quite a number of genetic anomalies with dominant effects on the tail have been described in the literature. Several of these genes, even those which appear to affect only the tail when heterozygous, have a drastic effect on the nervous system of homozygous embryos and are lethal before or at birth, as is true in this case. In most or all of the cases reported previously the involvement of the nervous system has been suggested to be dependent upon a prior effect of the gene upon the notochord or mesoderm organizer tissue.[1-5] The gene for looptail appears to present an exception on this point since development of the notochord and mesoderm in homozygous embryos is normal, at least as far as can be told from sections prepared by ordinary histological techniques. No histochemical tests have been attempted. At the pres-

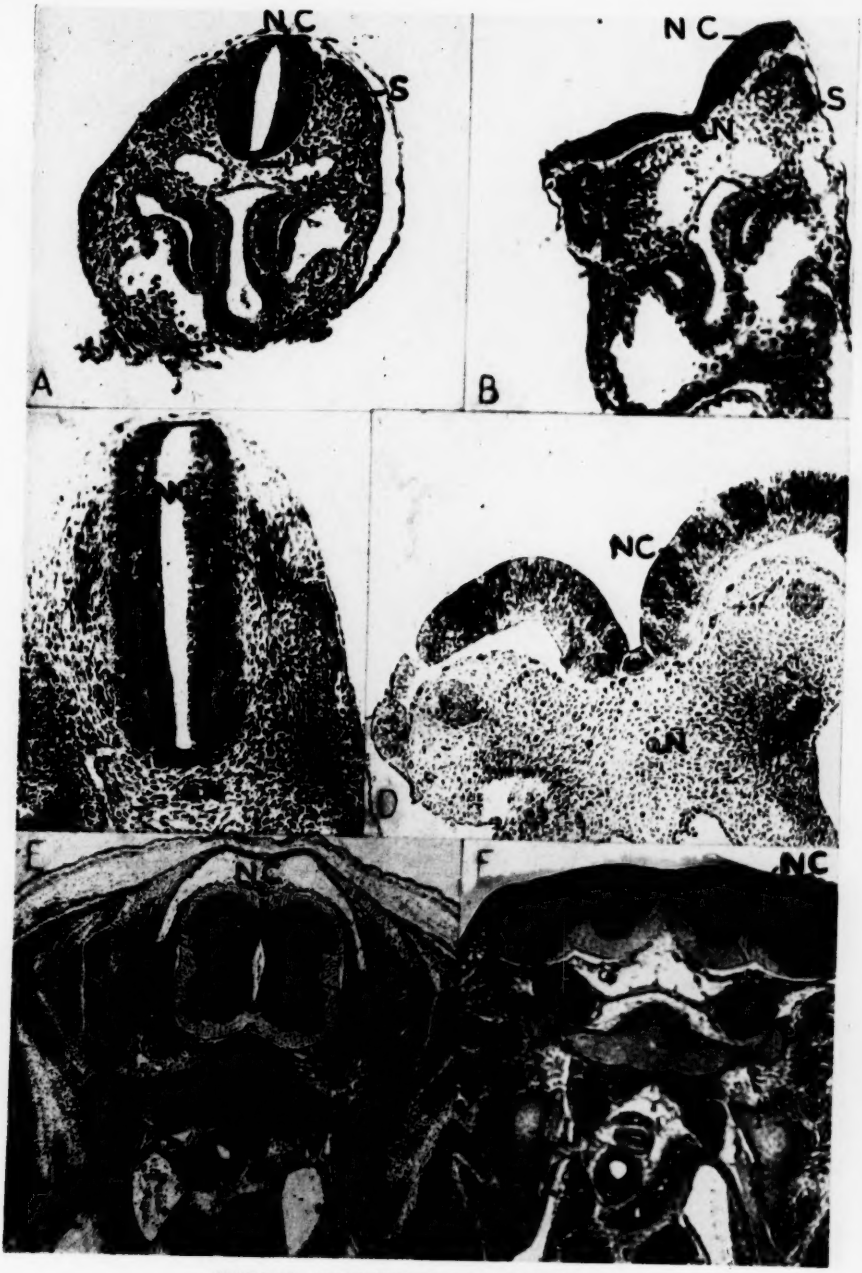
TABLE I. Number of mitotic figures in the region of the brain between eye and ear in normal and defective embryos

Age in days	Interval	No. of mitoses counted Defective	Normal	Total for region Defective	Normal	Av. ratio Def./Norm.
9	2	953	615	1906	1230	1.56
			605		1210	
10	11	2061	1348	22671	14828	
		1970	1426	21670	15686	1.45
11	9	4700	3137	42300	28233	
		4689		42201		1.50
15	9	3220	2402	28980	21618	1.34

TABLE II. Comparative numbers of mitoses in the spinal cords of normal and defective embryos

Age in days	No. of sections counted	No. of mitoses counted Defective	Normal	Ratio Def./Norm.
9	8	105	96	1.09
10	8	306	291	1.05
11	12	965	941	1.03
15*	8	18	2	9.

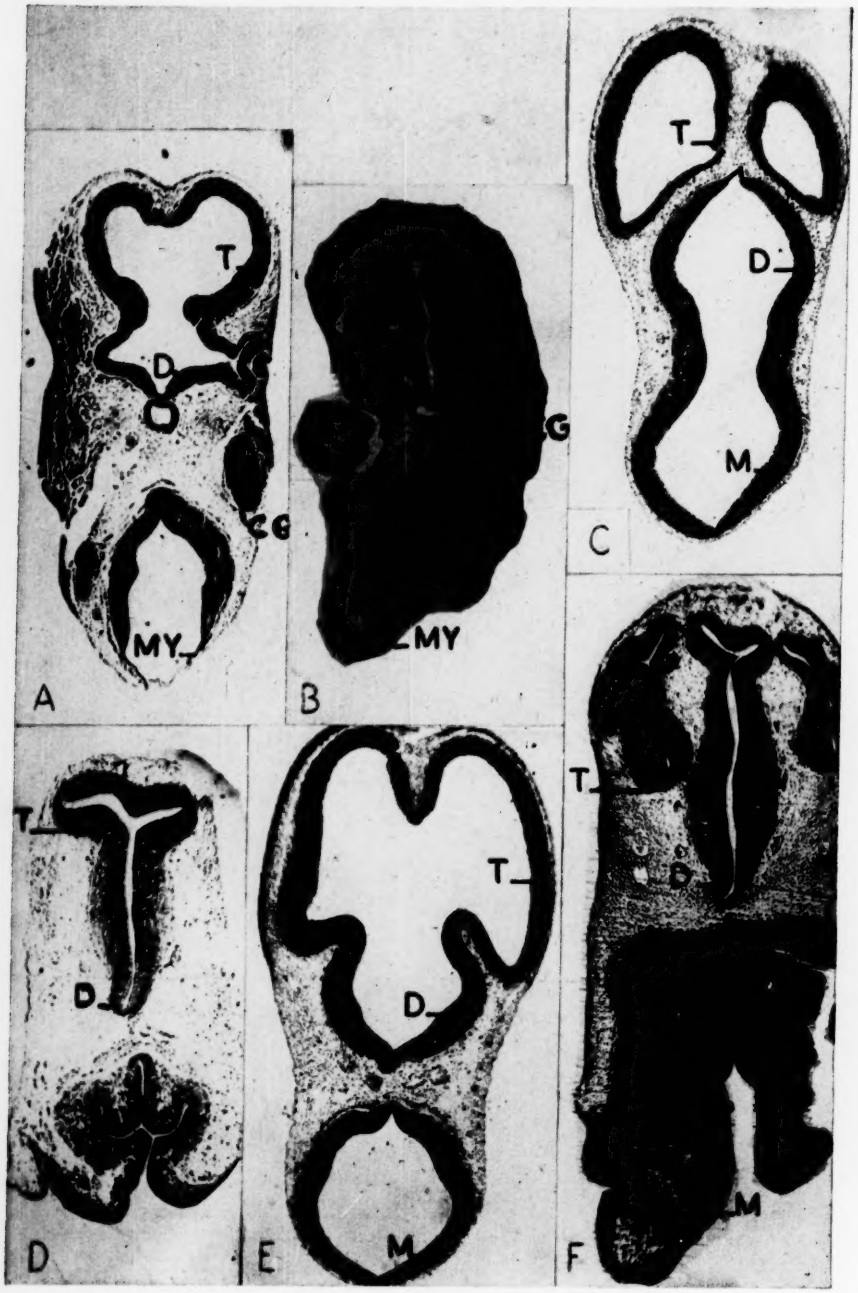
*In both normal and defective embryos the number of mitotic figures was so small that the ratio is probably not significant.



NORMAL NOTOCHORD DEVELOPMENT

Figure 12

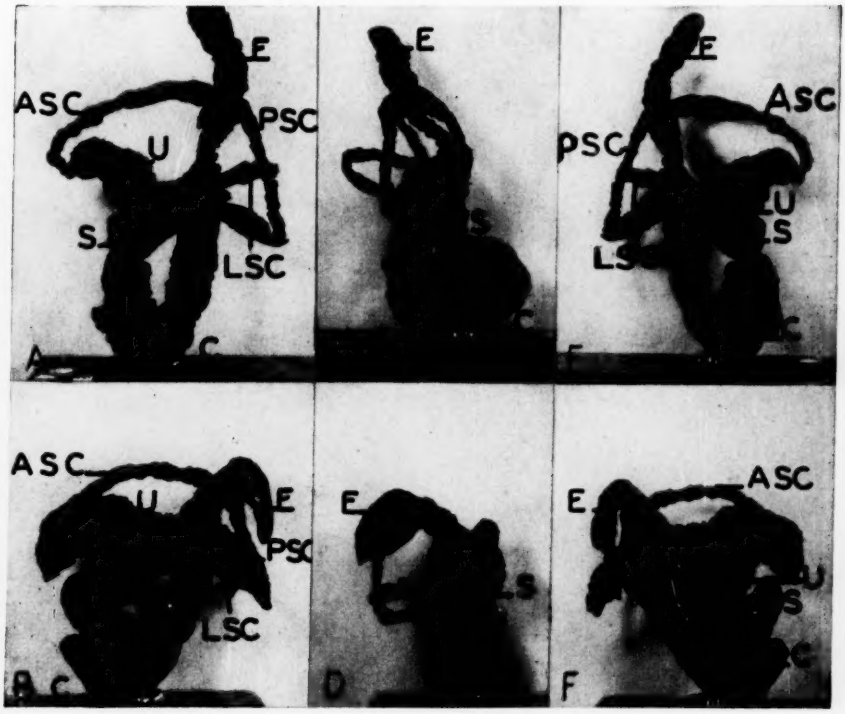
Cross Sections of normal—*A*, *C* and *E*; and defective—*B*, *D* and *F*, embryos. *A* and *B*—nine days (×100), *C* and *D*—ten days (×100), *E* and *F*—15 days (×40). (N — notochord, S — somite, V — vertebral arch, N C — spinal cord, S G — spinal ganglion).



DEVELOPMENT OF NERVOUS SYSTEM

Figure 13

Cross sections (×40) of normal (*A*) and defective (*B*) embryos at ten days. *C* and *E*— normal and *D* and *F* — defective embryos 11 days of age. (CG — cranial ganglion, D — diencephalon, M — mesencephalon, M Y — myelencephalon, T — telencephalon).



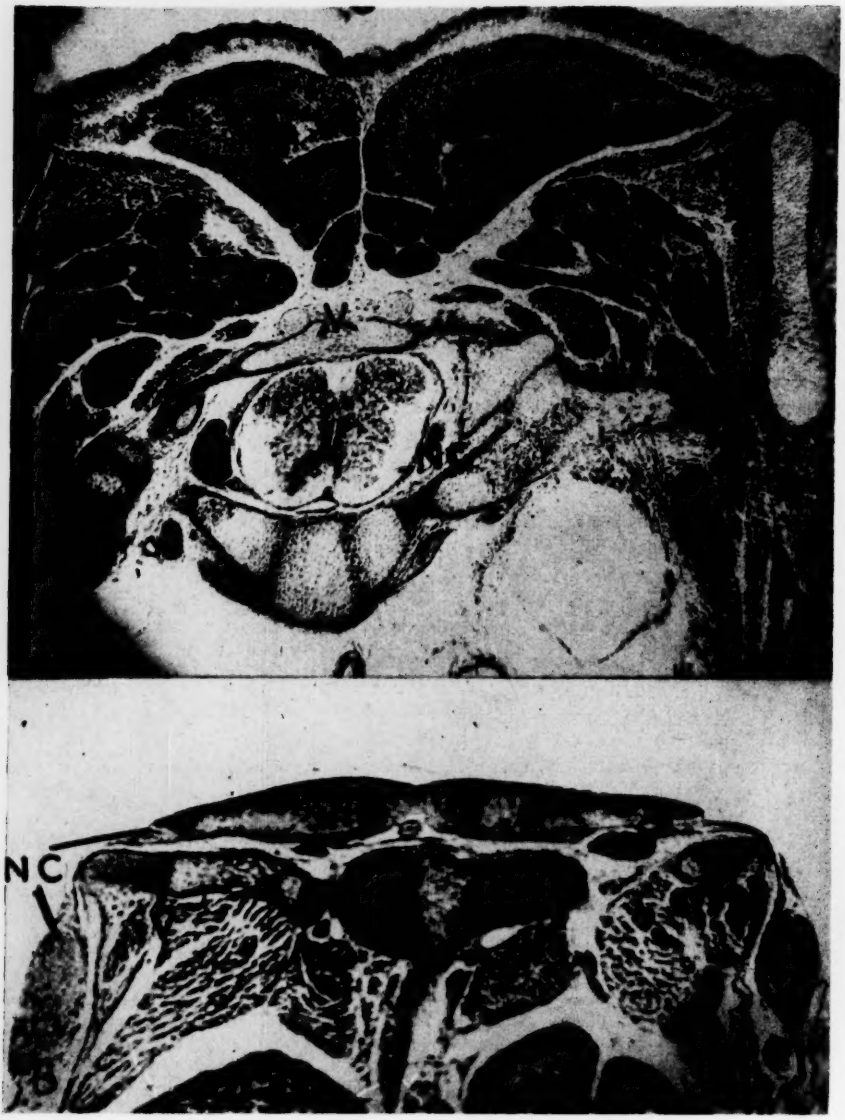
EARS OF NORMAL AND DEFECTIVE FIFTEEN-DAY EMBRYOS

Figure 14

Wax reconstructions of ear anlage in normal—*A*, *C* and *E*, and in homozygous looptail embryo—*B*, *D* and *F*. (A — anterior, P — posterior, L — lateral, S C— semicircular canals, C — cochlea, E — endolymphatic sac, S — sacculus, U — utriculur).

ent writing the only cytological difference which has been detected between normal and homozygous abnormal embryos is an increased number of mitoses in the region of the brain between the attachment of the optic stalk and the beginning of the auditory anlage. This area includes the major portion of the region of the brain which remains open in these homozygous embryos. The results to date may thus be interpreted to indicate a localized effect of the gene on this region of the developing nervous system.

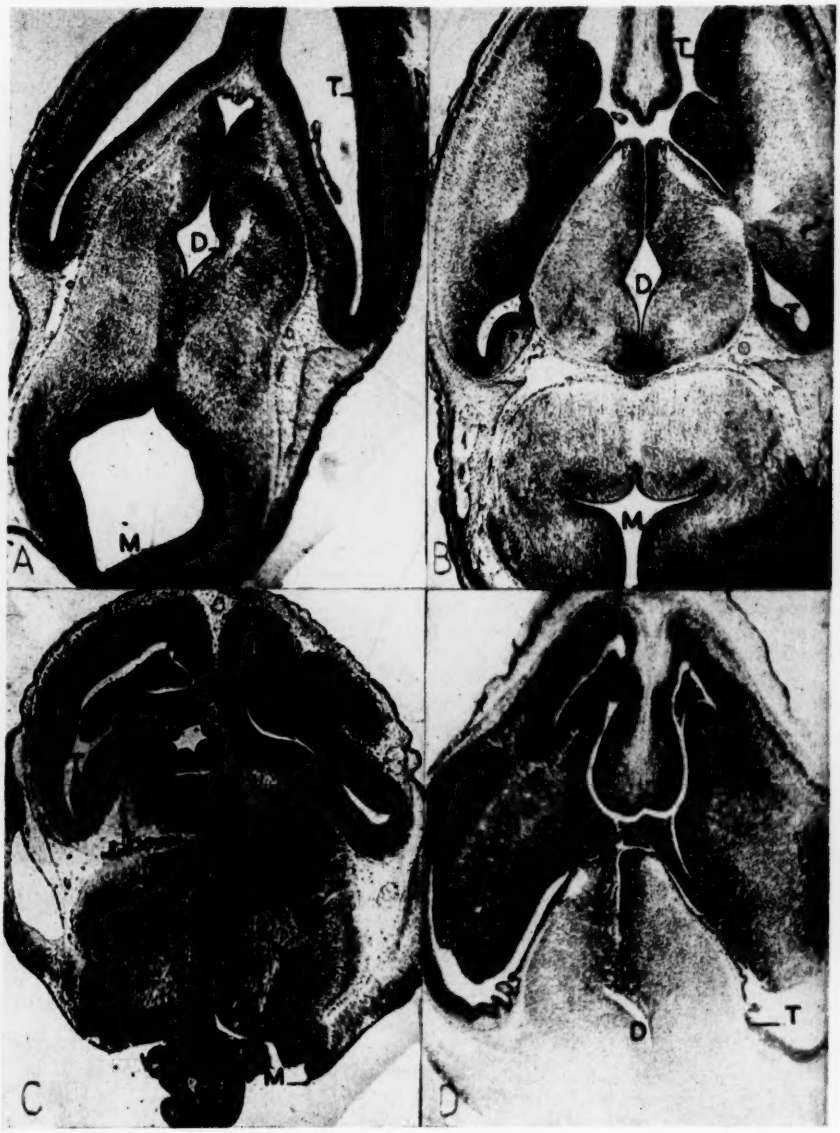
Hsu and Van Dyke[8] investigated the spina bifida and kinky tail in mouse embryos homozygous for "splotch" which die in utero,[12] and reported accelerated growth of the abnormal neural epithelium. They suggested, however, that the overgrowth was probably not the primary gene effect since early stages were retarded. It is interesting that they report accelerated growth at 13½ days and greater frequency of mitotic proliferation by 14½ days, while in looptailed mice mitotic divisions were more numerous at 9-10 days as well as later. According to Russell[11] the critical period for the production of spina bifida in mice by means of X-rays is the ninth day. Patten[10] has recently suggested that local overgrowth of the neural plate in early stages of development may be a factor in the production of some human anomalies. The conclusion was based on the examination of



VERTEBRAL ARCHES IN 2½ WEEK OLD EMBRYOS

Figure 15

A—cross section of closed cord, (N C), and vertebral arch, (V). *B*—open cord and vertebral arch piercing neural tissue. (×40).



NORMAL AND DEFECTIVE BRAINS OF FIFTEEN-DAY EMBRYOS

Figure 16

Cross sections (×32) of heads of normal embryos—*A* and *B*, and of homozygous looptail—*C* and *D*. (D — diencephalon, M — mesencephalon, T — telencephalon) .

sections of human embryos in which the walls of the neural tube were folded as if they had grown too large for the space in the head which they were destined to occupy. Patten points out that acceleration of growth rather than arrest of development may be involved in congenital anomalies. This would certainly seem to be true in the case of the homozygous looptail mouse embryos.

Since the spinal cord frequently, though not invariably, also fails to close in homozygous looptail mouse embryos, a difference in proliferative activity similar to that found in the brain might be expected in the cord area. No such difference was found. Perhaps those embryos in which the cord remains open differ from those in which it closes in that the area where closure first begins, that is in the region of the second and third somites, is within the localized region affected by the gene and if this area fails to close then the entire cord remains open. If this area is not involved, perhaps due to a variation in timing, then mid and hindbrain only are affected. No evidence exists at present however to support this suggestion. An analysis of the effect of a single dose of the gene for looptail also waits upon further study.

Summary and Conclusions

1. An analysis of the development of mouse embryos homozygous for the gene for looptail has been made by a study of histological sections of affected individuals and their normal litter mates from 8 to 18 days of embryological development.

2. Affected homozygotes, that is those showing open nervous systems, were not detectable until the middle of the ninth day at the time when closure of the neural folds normally occurs. Histochemical studies have not been made however, and might perhaps reveal earlier differences.

3. The notochord and mesodermal structures develop normally in affected embryos and it has therefore been concluded that the nervous system effect is not secondary to an effect of the gene on organizer tissue.

4. Differentiation of white and gray matter of brain and chord, development of the optic and olfactory anlage and of cranial and spinal ganglia and nerves also seem normal. The same applies to the development of the ear and of the kidney and endocrine glands.

5. The only difference detected to date between the abnormal embryos and their normal littermates, aside from the open nervous system and vertebral arches, consists of an increased proliferation of nervous tissue in the former. This was detected by counting the number of mitotic figures in the region of the brain between the attachment of the optic stalk and the level of the most anterior sections of the auditory anlage. The region counted included the greater part of that section of the brain which remains open in affected mice, namely the hindbrain and part of the mesencephalon.

6. Since similar counts of mitoses in areas of the cord showed no consistent difference between normal and affected individuals the results are interpreted as indicating a localized effect of the homozygous genes.

7. No attempt has been made to analyze the effect of the gene in the heterozygous condition. Penetrance, as regards the effect on the tail, and irrespective of the effect on the nervous system evidenced by wobbly behavior of the head, has been estimated at approximately 71 percent.

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'LYSENKOISM' IN WASHINGTON

American Scientists Find Fearful Parallel in Russia For Weeks' Approach to U. S. Bureau of Standards

Lowell Mellett

WHEN American scientists discuss among themselves the dispute between Secretary of Commerce Weeks and Dr. Allen V. Astin, head of the United States Bureau of Standards, there is one word they are apt to use. The word is "Lysenkoism!" It has come to have two meanings. One is a theory of genetics, the science of plant and animal breeding. Briefly stated, it is the theory that acquired characteristics in plants and animals can be inherited, a theory not much accepted among non-Soviet scientists. The other and in a way more important meaning is the theory that scientific thinking should conform to political thinking—should follow the party line.

Secretary Weeks, some of our scientists are saying, is endeavoring to introduce Lysenkoism into American science.

The word came into use some time after the fall of 1948 when a volume was published in Moscow under the title, "The Situation in Biological Science." It was a verbatim report of the proceedings of a meeting of the All-Lenin Academy of Agricultural Sciences. The "proceedings" were slightly fantastic, having some of the flavor of some Senate and House committee hearings. More or less on trial were all the Russian eggheads who held to the same concepts and principles of genetics as their professional colleagues engaged in "capitalist" science. The chief inquisitor was a rising young "Marxist scientist," Trofim Lysenko, a man of fanatical enthusiasm but no great learning. "To talk to Lysenko," according to one western scientist who had that experience, "was like trying to explain differential calculus to a man who did not know the multiplication table."

The young man, however, apparently appealed to the Kremlin and in 1941 he replaced Dr. Nicolai Vavilov, distinguished abroad as well as at home, as president of the All-Lenin Academy. Vavilov disappeared, and was reported to have died in 1943 in Siberia because he had lost his food-ration card.

On the final day of the 1948 meeting, Lysenko summed up the proceedings, saying, "I consider it my duty to make the following statement: The question is asked in one of the notes handed me, 'What is the attitude of the Central Committee of the Party to my report?' I answer: The Central Committee of the Party examined my report and approved it."

That, say American scientists, was the first time in the present century that any scientific group had frankly substituted political policy for the usual processes of science. But it seemed proper to the official party paper, Pravda, which in criticism of another group, said: "The presidium of the Academy of Sciences and the Bureau of the Biological Department forgot the most important principle of any science—the party principle."

The party principle, say the scientists, that Secretary Weeks has sought to apply in the Astin case is the new principle that it is not the customer who is always right, but any man who has something to sell. They say that will not stand scientific analysis, no matter how anxious Mr. Weeks may be to prove his friendship for business.

And they say, no matter how many Communists or alleged Communists the new administration may "drive out of Washington," the Nation will not be the gainer if at the same time it welcomes Communist practices. But, they say, if it is to be like that, the fact should be made clear.

The Washington Evening Star,
April 18, 1953. (Reprinted by permission)

GENETICS OF THE PAPAYA

W. B. Storey*

THE papaya (*Carica papaya* L., family Caricaceae) is a polygamous species of small, semi-woody dicotyledonous tree which is cultivated extensively in the tropics for its edible melon-like fruit.

The papaya has long been important to peoples of the tropics because it is one of the few plants which fruit throughout the year. In this regard, it differs from most other tropical fruit plants which, like fruit plants of the temperate zones, have fairly well-defined, comparatively short bearing seasons. Papaya trees, when well grown under favorable environmental conditions, commence producing ripe fruit at about a year of age. They may live for 25 years or longer, bearing continuously, with one or more fruit in each leaf axil.

Higgins[2] in 1916 published a paper in *The Journal of Heredity* on "Growing Melons in Trees" in which he remarked on the increasing importance of the papaya as a tropical fruit crop and on the need for breeding work to further enhance its value. At the time, the papaya was just emerging from the status of a home garden crop to that of a commercial orchard crop in Hawaii. In ensuing years, it has become an important orchard crop in Hawaii, South Africa, India, tropical America, and other places in the tropics. Its culture is being undertaken on a commercial basis in the United States in Florida, the lower Rio Grande Valley of Texas, and southern California.

In his paper, Higgins discussed tree forms, peculiarities of sex, problems of breeding, and ideals in breeding. Nothing was known of papaya genetics at that time. Some knowledge of papaya genetics has been gained in recent years, especially as regards tree forms and sex determination, from studies carried on principally in Hawaii, South Africa, and India.

In view of continuing interest in papaya genetics, cytology, and breeding at research centers in a number of tropical countries, the writer submits this paper as a summary report on the present status of some of the work. Some of the findings of genetics, particularly those which relate to sex segregations, are now being applied to seed production and to field planting in Hawaii and possibly elsewhere.

Tree Forms

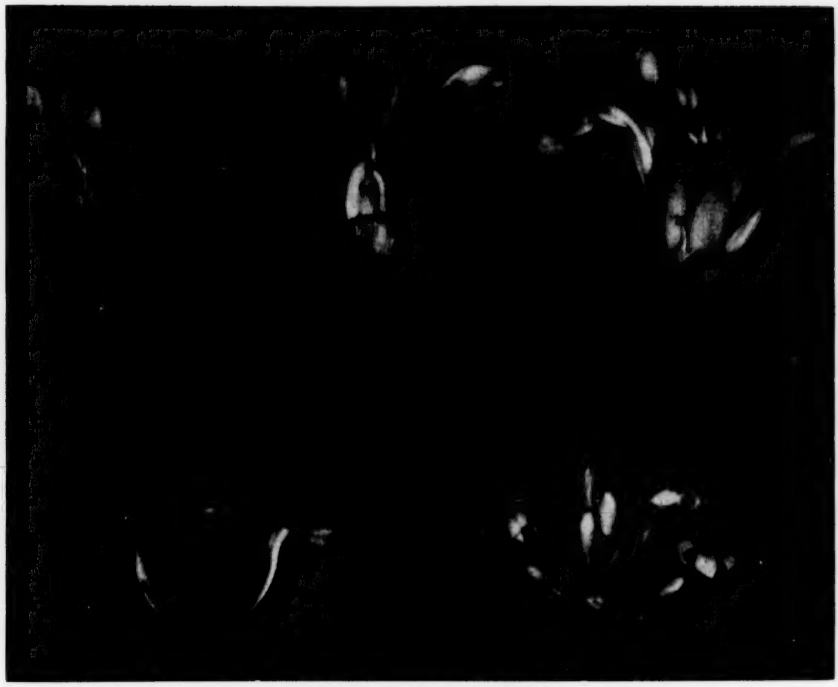
Papaya flowers are staminate, hermaphrodite, and pistillate (Figure 17). Teratological forms of hermaphrodite flowers are frequently encountered in which one or more of the stamens has become carpelloid and adnate to the pistil (Figure 17*B*), giving rise to deformed fruits.

Descriptions of tree forms, or "sex forms," as they are frequently called, have been based on floral composition which may consist of various admixtures of normal and teratological forms of flowers on long, many-flowered, or short, few-flowered inflorescences. To the present, at least 15 "sex forms" have been described in the literature.[2,3,11] It has been pointed out by Hofmeyr[4] and Storey,[19] however, that each of the tree forms properly belongs to one of three basic sex types: (1) staminate, or "male"; (2) hermaphrodite; (3) pistillate, or "female," and that all of the forms, excepting the female, which is a stable monotypic form, are males and hermaphrodites whose flowers vary in sex expression under different environmental conditions.

Workers with the papaya are now more or less generally agreed that trees having long, pendulous, many-flowered cymose inflorescences are male forms. Such trees may bear staminate flowers only throughout the year (Figure 18*A*), or they may be female-fertile at certain times of the year, when they produce few to many bisexual flowers which develop into fruits (Figure 18*B*).

Trees with peduncles only a few inches long bearing relatively few flowers, usually no more than 15, most of which are bisexual, are regarded to be hermaphrodite forms (Figure 18*C*). Most hermaphrodite trees are fruitful

*Horticulturist, University of Hawaii, Agricultural Experiment Station. Published with the approval of the Director as Technical Paper No. 264 of the University of Hawaii, Agricultural Experiment Station, Honolulu 14, Hawaii.



PAPAYA FLOWER TYPES

Figure 17

A—Ten-stamened elongata hermaphrodite; *B*—Elongata with carpelloid stamens; *C*—Five-stamened pentandria hermaphrodite; *D*—Pistillate; *E*—Staminate.

throughout the year, but some may become completely female-sterile at certain times of the year (Figure 19*A*).

Occasional mention is made in the literature of monoecious papaya trees. No truly monoecious form, that is, one bearing staminate and pistillate flowers either on the same peduncle or on separate peduncles, has ever been recorded in Hawaii. Andromonoecious forms are common, and the writer and other workers have tended to classify them as "hermaphrodites." It is probable that andromonoecious forms are what are referred to where the term "monoecious" is used in the literature.

The principal character which distinguishes males from hermaphrodites is length of the primary peduncle. Similar admixtures of normal and teratological flowers occur in variant forms of both kinds of trees. Seasonal sex reversal takes place both in males and in hermaphrodites at the same time and in the same direction. The only variant form which does not yet seem to have been recorded is a continuously female-fertile male comparable to the continuously fruitful hermaphrodite.

Trees which bear flowers that are characteristically and constantly pistillate on short peduncles, usually with fewer than five flowers, are regarded to be females (Figure 18*D*). As previously noted, the female tree is remarkably stable and monotypic in form. With a few possible exceptions noted by Hofmeyr[6] in a single progeny, it is not known to vary in sex expression in response to environmental influences.

Sex Determination

The three sex forms in papaya, as well as the numerous variant forms, are indistinguishable from one another in vegetative characters. The sex of an individual plant can be determined only from its flowers, which begin to appear six months or more after germination of the seed. Workers in a number of papaya-growing countries have carried on investigations to discover a sex-linked character in seeds or in juvenile vegetative characters of seedlings which might be useful in early separation of the sexes, but none has succeeded

PAPAYA TREE FORMS

Figure 18

A—Staminate; *B*—Seasonally female-fertile staminate; *C*—Hermaphrodite; *D*—Pistillate.

in finding anything of immediate practical value.

Genetical studies on sex determination which were carried on independently by Hofmeyr[4] in South Africa and by Storey[19] in Hawaii have shown that although the sex of a single individual may not be known, the sex phenotypes and their proportions may be predicted for progenies produced from controlled pollinations.

Sex segregations determined by Hofmeyr and by Storey from progenies which, at this

PAPAYA SEX MODIFICATION

Figure 19

A—Seasonally female-sterile hermaphrodite tree; *B*—Seasonally female-fertile staminate tree with high percentage of fruits deformed by stamen carpellody.

writing, have aggregated several tens of thousands of individuals are given in Table I.

Both workers concluded from the ratios given in Table I that sex in the papaya is determined by a single gene with three alleles, to which Hofmeyr[4] assigned the symbols: M_1, staminate; M_2, hermaphrodite; m, pistillate. Storey[18] reported all combinations of dominants, M_1M_1, M_1M_2, and M_2M_2, to be lethal to the zygotes. Males and hermaphrodites, therefore, are enforced sex heterozygotes, and their fruits contain 25 percent nonviable seeds. In this hypothesis, therefore, the genotypes of the sex types are: M_1m, male; M_2m, hermaphrodite; mm, female. The crosses and their progeny segregations corresponding to those in Table I, with the nonviable genotypes in parenthesis, are as follows:

1. $mm \times M_1m \longrightarrow 1\ mm : 1\ M_1m$
2. $mm \times M_2m \longrightarrow 1\ mm : 1\ M_2m$
3. M_1m selfed, and $M_1m \times M_1m \longrightarrow$ $1\ mm : 2\ M_1m : 1\ (M_1M_1)$
4. M_2m selfed, and $M_2m \times M_2m \longrightarrow$ $1\ mm : 2\ M_2m : 1\ (M_2M_2)$
5. $M_2m \times M_1m$, and $M_1m \times M_2m \longrightarrow$ $1\ mm : 1\ M_2m : 1\ M_1m : 1\ (M_1M_2)$

Since males and hermaphrodites are heterozygous for sex and females are homozygous, sex determination in the species has been regarded by some workers to be of the XY type.[1, 12]

Hofmeyr[6] inclines toward the genic balance hypothesis for sex determination in papaya. He has assigned an arbitrary value of 47 to the autosomal chromosome complement in favor of maleness, and values of $m = 25$, $M_1 = 21$, and $M_2 = 22$ to the sex chromosomes in favor of femaleness. Thus, the genotype mm has a value of 3 in favor of femaleness, M_1m has a value of 1 in favor of maleness, and M_2m has the threshold value of 0. The female is quite stable because its value of 3 places it well above the threshold value. Proximity to the threshold value is reflected by the sexual instability of males and hermaphrodites, whose sex expression may be influenced by modifying

TABLE I. Sex ratios resulting from cross- and self-pollinations of sex forms in *Carica papaya*

Pollination	Sex Ratios ♀♀	☿☿	♂♂
1. ♀ × ♂	1	0	1
2. ♀ × ☿	1	1	0
3. ♂ selfed, and ♂ × ♂	1	0	2
4. ☿ selfed, and ☿ × ☿	1	2	0
5. ☿ × ♂, and ♂ × ☿	1	1	1

factors which push the balance closer to or farther away from the threshold value. Although Hofmeyr[9,10] and van Elden[10] have done some work with colchicine-induced polyploids which tends to support the hypothesis, further investigation seems indicated, particularly on aneuploid plants, to test fully its validity.

The writer's more recent investigations have led him to believe that sex in papaya is determined not by a single gene but rather by a complex of genes which lie closely linked in differential segments occupying identical regions on the sex chromosomes. The sex-determining segments behave in heredity as if they were unit factors. What appear to be pleiotropic effects of unit factor alleles, such as differences in peduncle length, functionability of androecium and gynoecium, shape of ovary, size of fruit, and gross morphology of flower, may be attributed to the gene complexes resident in the differential segments.

Genes known, or surmised, to lie in the sex-determining segments are as follows:

Mp, male peduncle, i.e. the long, ramified, many-flowered cymose inflorescence of the male tree; *mp*, or +, the short, sparingly-branched few-flowered inflorescence of the female and the hermaphrodite.

l, zygotic lethal

sa, suppressor of the androecium

sg, suppressor of the gynoecium

C, hypothetical factor for suppression of crossing over in the region of the sex differentials.

The hypothetical sex genotypes are given below. The order of the genes in the sex-determining segments, however, has not actually been determined.

$$\delta = \frac{M_1}{m} = \frac{Mp \quad l \quad C \quad + \quad sg}{+ \quad + \quad + \quad sa \quad +}$$

$$\varphi = \frac{M_2}{m} = \frac{+ \quad l \quad C \quad + \quad sg}{+ \quad + \quad + \quad sa \quad +}$$

$$\varphi' = \frac{m}{m} = \frac{+ \quad + \quad + \quad sa \quad +}{+ \quad + \quad + \quad sa \quad +}$$

Length of peduncle (*Mp*, +), a secondary sex character, is the principal distinguishing difference between males and hermaphrodites. As noted preivously, tree forms with long inflorescences have come to be regarded as males regardless of floral composition. Short peduncle is a character common to hermaphrodites and females.

The zygotic lethal gene (*l*) is common to males and hermaphrodites, and is the factor which enforces heterozygosity on these sex forms. No sexually homozygous males and hermaphrodites and no lethal-gene-bearing females have been reported, indicating that no crossing over takes place between *l* and the other genes of the sex-determining region.

The symbol *C* is inserted to represent a factor, possibly a gene, but possibly also a chromosomal inversion, a deletion, or proximity of the sex segment to the kinetochore or to the end of the chromosome, which precludes crossing over between the other genes of the complex. The presence of such a factor is assumed because, to the best of the writer's knowledge, no cross-over types such as long-peduncled females, lethal-gene-bearing females, and non-lethal homozygous males and hermaphrodites have ever been found. One cross-over type, which might be called a "short-peduncled male," has been reported in the literature as a completely sterile form of hermaphrodite by some observers.[2,3]

Hofmeyr[6] has suggested that M_1 and M_2 may represent small inactivated or inert regions of the sex chromosomes, with the M_1 section slightly longer than the M_2 section. If vital genes are missing from these sections the M_1M_1, M_2M_2, and M_1M_2 genotypes would be rendered nonviable.

Overlapping chromosomal deletions may well be the cause of zygotic lethality, and the cause of suppression of crossing over as well. If this be the case, the functions of *l* and *C* could relate to a single chromosomal deficiency, rather than to two separate closely linked genes.

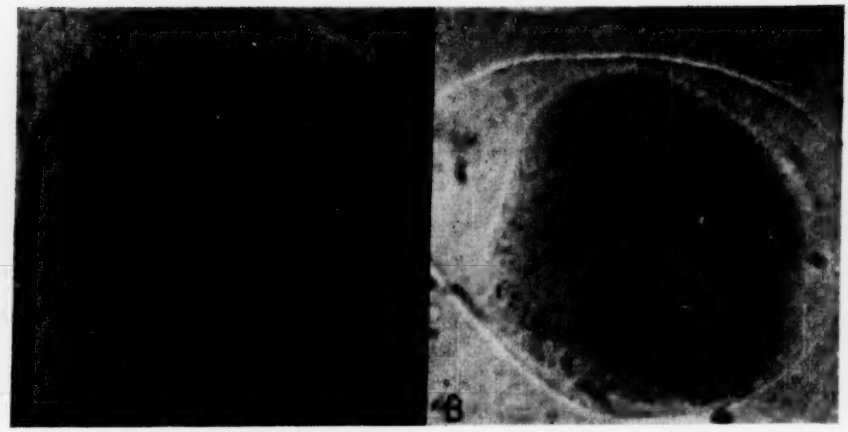
The gene *sa*, when homozygous, as in the female, effects complete suppression of the androecium. It is apparently not completely recessive to its normal allele, but may exert some influence on suppression of the androecium in males and hermaphrodites under certain environmental conditions, possibly through interaction with sex-modifying factors. Its influence on fruiting males and hermaphrodites is best observed in the cool months of the year, when there is a strong tendency for sex expression to shift in the direction of femaleness.

The gene *sg*, when homozygous, as in the completely female-sterile male, effects complete suppression of the gynoecium. Most forms of males and hermaphrodites seem to be heterozygous for the gene, and their sex expression tends to be forced in the direction of maleness in the warm months of the year. Constantly fruitful hermaphrodites probably are homozygous for the normal allele for gynoecium development.

Since the sex-determining complexes are inherited as if they were unit factors, the writer has continued to use the symbols *M*, M^h, and *m*, for convenience, but to represent the male, hermaphrodite, and female differential segments respectively rather than to represent unit sex-determining factors.

Modifiers of Sex Expression

There are two independent sets of factors which may modify sex expression in males and hermaphrodites under certain environmental conditions. One set causes seasonal shifts from



PAPAYA CHROMOSOMES IN POLLEN MOTHER CELLS

Figure 20

A—Meiotic premetaphase I ($n = 9$); *B*—Precocious separation of one pair at Anaphase I, ($\times$ 2250).

female fertility to sterility and back again. The other set causes stamens to become carpelloid, usually with fusion to the pistil. It is largely the expressions of these sets of factors, either singly or in combination, which seem to have been the basis for the concept of 15 sex forms in papaya.

Female-sterility behaves as a sex-limited character, and is expressed only in males and hermaphrodites (Figures 18*A*, 18*B*, and 19 *A*). The stability of pistillate flower production by the female has already been remarked upon. Nevertheless, the factors for female-sterility may be carried by the female and passed on to the offspring.

Carpellody of stamens is perforce limited to males and hermaphrodites, although the causative factors, like those of female-sterility, may be carried by females. Trees free of the factors produce normal fruits in all seasons of the year. Trees carrying the factors produce mixtures of normal and teratological fruits under the proper stimuli (Figure 19*B*).

In order for a male or a hermaphrodite tree to undergo a change in sexual functionability, either female-sterility or stamen carpellody, the potentiality to do so must be inherent in its genotype. Even so, the potentiality does not come to expression unless proper physiological conditions obtain.

The genetical aspects of female-sterility and stamen carpellody have not yet been worked out, but studies in progress indicate that in each case the potentiality for character expression depends upon several pairs of genes rather than a single pair.

Sex Chromosome Studies

The papaya has nine pairs of chromosomes (Figure 20*A*).

A number of cytological studies have been conducted on papaya to determine whether it, like numerous other species of plants which have distinct sex forms, has a heteromorphic pair of sex chromosomes.

Meurman,[14] Suguira,[20] Lindsay,[13] van Elden,[1] Hofmyer,[4] and Storey[19] reported variously from earlier studies that they could discern no heteromorphism in a chromosome pair either among somatic chromosomes or among chromosomes in various stages of meiosis. Kumar, Abraham, and Srinivasan[12] have reported from fairly recent studies that one pair of chromosomes separates precociously at anaphase I of meiosis in males and hermaphrodites (Figure 20*B*). They observed no such precicious anaphasic separation in meiosis in the female, and have remarked that the difference in behavior is in keeping with what has been observed in other species of plants which have heteromorphic sex chromosomes. They noted also, however, that they could detect no morphological differences between disjoining homologues.

The writer has subsequently examined numerous dividing pollen mother cells from male and hermaphrodite trees. Precocious anaphasic separation was observed to occur with high frequency, but it was not observed in every cell at anaphase.

Precocious anaphasic separation may indeed be a feature which serves to set the sex chromosomes apart from the autosomes. Further

study seems indicated, however, to determine with certainty which one of the nine pairs behaves in this way, and whether a means may be discovered to distinguish the disjoining sister chromosomes from each other.

Qualitative Characters

Mutant papaya genes which have been studied are:

a, albino plant, recessive to normal green plant.

d, dwarf plant, recessive to normal tall plant; characterized by early excessive branching.[9]

dp, diminutive plant; recessive to large plant; characterized by short, slender trunk, small leaves with short, slender petioles, and small flowers and fruits.

cp, crippled leaf, recessive to normal flat leaf; appearance similar to severe crippling by papaya mosaic virus.

rg, rugose leaf, recessive to normal smooth leaf; characterized by upward puckering of blade areas between veinlets and by various degress of curling of the margins.

w, wavy leaf, recessive to normal flat-bladed leaf.

r, red flesh of fruit, recessive to yellow flesh.[4]

Y, yellow flower color, dominant to white.[4]

P, purple stem and petioles, dominant to green[4]; intensity of pigmentation may be affected by modifying factors not yet analyzed.

B, gray seed coat, dominant to black seed coat.[5]

Studies of mutant genes in the papaya have revolved largely around attempts to find a sex-linked vegetative character which might be useful in separating the sexes at an early seedling stage. All of the genes listed above, however, excepting *Y*, *P*, and *B*, are inherited as autosomals.

Hofmyer[6] reported *Y* and *P* to be sex-linked with the following approximate relationship: *M*₁ *25 Y 16 p*. The reported cross-over values have been confirmed by the writer in material given him by Hofmyer for testing. Obviously the linkage between sex and flower color is of no practical value, since it cannot be applied until time of flowering, when floral morphology itself is a better means of determining sex. The cross-over value of approximately 41 percent between sex and stem color so nearly approximates 50 percent, at which assortment is virtually random, that it confers no particular advantage in selecting out sex types for commercial orchard planting.

Hofmyer[5] has reported sex and seed coat color to be linked with 31.1 ± 4.5 percent crossing over. The position of the seed color gene in relation to the other genes on the sex chromosome seems not to have been determined. Its position closer to the sex locus than *P* might make it of some value in selecting higher proportions of preferred sex types prior to planting. The character has not been observed in Hawaii.

Crossing over occurs freely between the sex locus and the *P* and *Y* genes, and probably the *B* gene also, regardless of the sex of the individual tree. This freedom of crossing over lends support to the hypothesis that the sex determining factors are confined to small differential regions on the sex chromosome within which crossing over is precluded.

Quantitative Characters

Basic fruit shape is determined by the sex of the tree, or, more specifically, by the flower type from which the fruit develops.[16] Shape may be modified somewhat by quantitatively inherited factors. Thus fruits from hermaphrodite flowers may range in shape from long cylindrical through short pyriform to nearly spherical, and fruits from pistillate flowers may range from oblate spheroidal to ovoid. Varietal differences in fruit shape for a given sex type of tree seem to relate largely to such factors.

Fruit size of an F_1 hybrid progeny in terms of volume tends to lie near the arithmetic mean of its inbred parental lines, and, in the F_2, to cover the range from one parental extreme to the other. The same distribution is found in fruit weight. It has been found that fruits from female trees in an inbred gynodioecious variety average about 1.25 times as heavy as fruits from elongata hermaphrodite flowers on hermaphrodite trees, suggesting the presence of a weight difference factor in some way correlated with sex.

Tree stature is determined largely by length of internode, which is influenced by multiple factors.[15] Studies in progress indicate that approximately equal numbers of new leaves are produced in a given period of time regardless of variety, so that final differences in stature relate to internode length rather than to numbers of nodes produced. Exceptions to quantitative inheritance of stature are the occurrence of dwarf plants reported by Hofmyer[9] to be due to a recesisve gene *d*, and of diminutive plants found by the writer to be due to a recessive gene *dp*. In these forms, the entire plant is dwarfed. Where internode length alone is concerned, there is no reduction in size of fruit, trunk girth, length of petiole, size of blade, or other plant characters.

A recently completed study by Nakasone[15] has shown that height of initial flowering is determined by two independent sets of multiple factors: (1) length of internode; (2) number of nodes from the cotyledonary node to the first flowering node. Time from seed germination to flowering, however, is highly correlated with number of nodes, so that the earliest flowering plants, which usually are also the lowest flowering plants, generally have the fewest nodes to the first flower. Internode length, although it affects the height of initial flowering slightly, has no apparent effect on earliness of flowering.

Practical Applications

The papaya is raised solely from seed for commercial planting, vegetative propagation of desirable types having been found uneconomical to the present. The plants commence to flower at an age of four to six months and to produce ripe fruits at an age of 9 to 14 months, depending on variety. Most plantations cannot be maintained in production economically for more than two or three crop years, so maintenance of a supply of seed of desirable commercial types assumes importance.

In earlier years, most varieties in cultivation in Hawaii were dioecious, and a practical difficulty from the standpoint of the grower was the fact that approximately 50 percent of the trees from a lot of seed were males, and therefore useless. Only a few males, no more than 10 percent, are needed to pollinate all of the female trees in an orchard planting. Furthermore, the grower had no way of distinguishing the sex of one seedling from another until they began to flower. The difficulty has been largely resolved by the development and use of gynodioecious varieties such as *Solo*. Originally a polygamous, highly variable variety, *Solo* has been developed into a fairly uniform gynodioecious variety, with every tree a bearing tree, by restricting pollination to the selfing or intercrossing of hermaphrodites for seed production.

With the basic pattern of sex determination known, it now seems a comparatively simple matter to convert any desirable dioecious variety to gynodioecism, with complete elimination of males, by initially pollinating the females with good types of hermaphrodites, followed by back-crossing until the desired varietal characteristics have been restored.

In Hawaii in recent years, a strong market preference has developed for the pyriform fruit which develops from the elongata flowers of hermaphrodite trees of the *Solo* variety. Seeds of selfed hermaphrodites yield only two hermaphrodites to one female, however, a ratio which the grower considers too high in females in orchard planting. With no means of separating individual seedlings according to sex, but knowing the expected proportion of sex types, the grower can reduce the proportion of females to hermaphrodites in his final orchard stand by planting two or three seedlings to a place, thinning to a single tree as soon as the sexes can be determined from the flowers. With two trees to a place, the proportion of females may be reduced to one out of every nine trees in the final stand, and with three trees it may be reduced to one out of 27 trees. This procedure has been adopted by numerous growers in Hawaii.

If dioecious varieties are adhered to, as they are in many places outside of Hawaii, the same thinning procedure may be followed. As mentioned earlier about 10 percent males in a planting is considered adequate for thorough pollination of the females. By planting three seedlings to a place, males may be reduced to about 12.5 percent of the final stand.

It is perhaps worth remarking that the yield of marketable fruit per acre has increased 114 percent in Hawaii since the first genetical studies were initiated in 1936. This has been accomplished largely through the breeding program of the Hawaii Agricultural Experiment Station, with careful selection of desirable types, and elimination of heritable nonproductive forms. The effects of improved cultural practices and of better methods of insect and disease control can in no wise be discounted, of course.

Conclusion

Higgins[2] listed the following as ideals in breeding the papaya: (1) vigor of tree; (2) early and low fruiting; (3) freedom from the branching habit; (4) productivity but not excessive bearing; (5) hermaphroditism; (6) suitable fruit size; (7) yield of papain; (8) uniformity of shape; (9) uniformity of ripening; (10) coloring before softening; (11) color of flesh, chrome yellow preferred; (12) easily separable placenta; (13) good flavor; (14) good keeping qualities. These ideals remain essentially unchanged today, and to them have been added thick flesh, firm flesh of good texture, small ovarial cavity, and regularity of ovarial cavity for ease in removing the seeds. Some of these ideals have been attained and others are near attainment, but some will

be attained only through an intelligent—and perhaps protracted—breeding program.

Progress in papaya breeding in the past has been hampered by a large number of popular misconceptions of sex forms and breeding behavior, and by lack of knowledge of the genetics of the species. As the genetics becomes better known it seems destined to play an important role in furthering breeding programs for improving the papaya.

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THREE JAPANESE PEDIGREES OF TYPICAL BRACHYDACTYLY

TAKU KOMAI*

OF all kinds of hereditary malformations of human extremities, the typical brachydactyly is characterized by the rudimentary state of the middle phalanges of *all* fingers and toes. The inheritance of this malformation in an American family was first reported by Farabee.[6] He concluded that the anomaly was due to a dominant gene. This was the first demonstration of dominant Mendelian inheritance in man. Farabee's study was soon followed by Drinkwater's[4] relating to an English kindred. This study yielded the same conclusion as Farabee's on the mode of inheritance of this characteristic. Six years later, Drinkwater[5] reported another English kindred with similar malformations of the digits. He was able to trace the pedigree to a member of Farabee's kindred who had migrated from America to England. These studies have become famous, since many genetic textbooks refer to these pedigrees.

Typical brachydactyly is by no means common. Lewis[11] refers to only two pedigrees, besides those of Farabee and Drinkwater—a French pedigree by Mercier[12] and a German pedigree by Hasselwanter.[9] Besides, there is an old pedigree reported by Webb[15] which undoubtedly concerns the same type of deformity as cited by Farabee and Drinkwater. Gilmore's pedigree[8] also seems to be of the same type of abnormality, although the patients have defective nails in addition to the digital malformation. Walter[14] gives a large pedigree of brachydactylous people found among mountaineers in West Virginia. However, this may represent a genetically different sort of abnormality, since only a few (not necessarily all) fingers and toes are said to be affected. The more recent kindred recorded by Iltis[10] closely resembles Drinkwater's in the manifestation of the deformity.

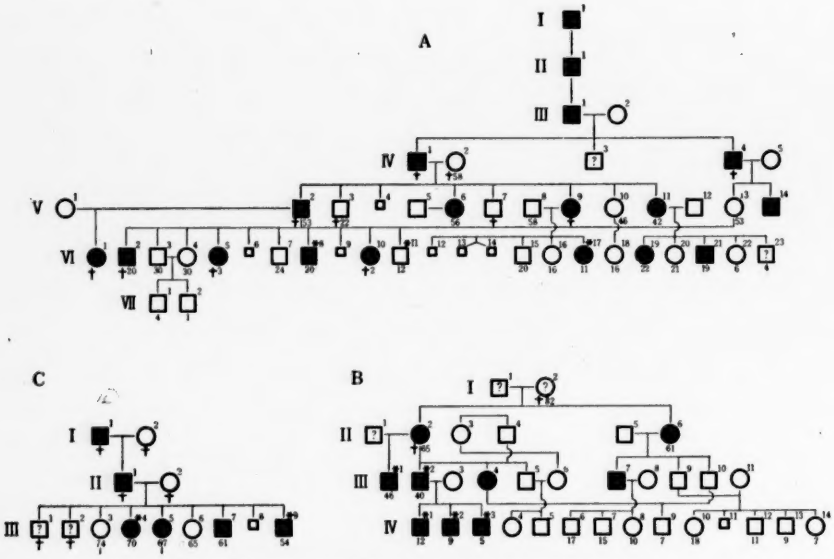
In Japanese literature, so far as the writer has been able to ascertain, no case of typical brachydactyly has been recorded until recently. It was only a few years ago that three kindreds were discovered. The wrtier has carefully investigated two of these kindreds, and confirmed both of them to be examples of typical brachydactyly. He was unable to study the third kindred, but there is no doubt that this also is typical. These three kindreds are residents of districts rather far removed from one another. Moreover, they show differences in the details of the deformity. Therefore, it seems rasonable to assume they are of entirely different lineage.

In pursuing these studies, the writer experienced rather serious difficulties by the lack of cooperation from the patients' relatives, and he often had to be content with indirect information when first-hand knowledge was desired. However, thanks to the effort of his friends, the information necessary for this report was obtained.

Kindred A

Most of the members of this kindred reside in Nagano Prefecture in the vicinity of Matumoto. This kindred was discovered by Mr. H. Aoki, a student at the Agricultural College in Tokyo, who is a native of a village near the home of this kindred. Last summer, the writer, with Mr. Aoki and his former teacher at Matumoto High School, Mr. R. Yamazaki, visited this place, and examined one of the individuals, (VI-8, Figure 21). The village office register and neighbors of the family in question also supplied information for the pedigree. Mr. Yamazaki visited a woman in this kindred (V-9), and took photographs and

*National Institute of Genetics, Misima, Sizouka-Ken, Japan. Contribution No. 32 of the Institute of Genetics. The writer was generously assisted by Mr. R. Yamazaki and Mr. H. Aoki in working out Kindred A, and by Mr. T. Akao for Kindred B. The information relating to Kindred C, he owes entirely to Dr. G. Yasuzumi and his associates.



PEDIGREE OF TYPICAL BRACHYDACTYLY

Figure 21

Three pedigrees of typical brachydactyly in the Japanese population. Numerals below symbols designate the age of the individual; a cross beneath the symbol indicates dead individuals; an asterisk indicates persons examined by the writer or some other investigator.

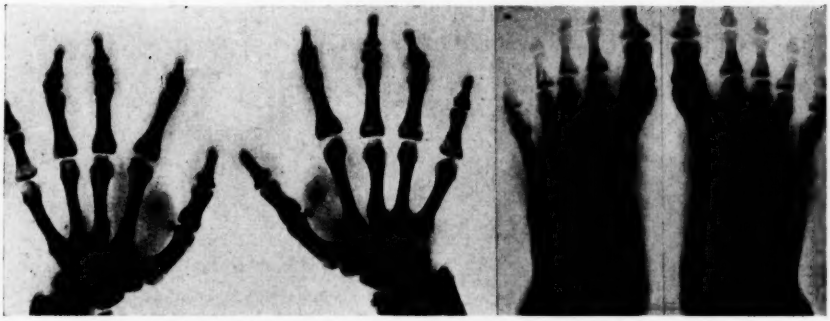
X-ray pictures of the brachydactylous hands and feet of one of her daughters (VI-17).

According to the register, III-1 was a man born about 1830. An old neighbor of the family who gave us valuable information of the kindred, remembered this man to have been short-fingered. The old neighbor also told us that III-1 was of the third generation of this short-fingered lineage. III-1 had, besides some normal children, two sons (IV-1 and IV-4), who were short-fingered. IV-1 was born in 1867, married a normal woman (IV-2) and died in 1925. This couple had four sons and four daughters. Of the sons, one died in infancy, and of the remaining three one (V-2) was abnormal. Of the daughters, one was normal, and three (V-6, V-9, V-11) were abnormal. IV-4 left his native village, and settled in Gumma Prefecture, and married. He had a son (V-14) who had the malformation.

V-2 married twice. The first wife (V-1) was feeble-minded; she gave birth to a brachydactylous girl (VI-1) who died in infancy. The second wife (V-13) was his cousin; the couple produced a large family consisting of seven sons and three daughters. Except for two sons who died in infancy, most of this generation are alive. Of the sons three are normal and two (VI-2, VI-8) are abnormal. The elder son (VI-2) committed suicide from worry about his deformity, when he was about to receive a physical examination for conscription. The other brachydactylous brother (VI-8) was interviewed by the writer. Of the three daughters, two (VI-5, VI-10) died in infancy, but they were known to have short fingers. V-9 married a man living in Matumoto. This couple had six children, three having died in infancy. Of the living children, a son and a daughter are normal, while the last child, a girl of eleven (VI-17) has brachydactylous hands and feet. V-11, another brachydactylous woman of the fifth generation, married a man living in Aiti Prefecture. They have three daughters and two sons, of which a girl (VI-19) and a boy (VI-21) are abnormal.

Description of the Anomaly of Kindred A

K. H. (VI-8) was interviewed by the writer. He is a man twenty years old, looks robust, with a stature of 153 cm. (The average stature of Japanese men being about 160 mm.) His hands look broad and thick; the metacarpal region looks normal. The fingers are short, and apparently have only two phalanges, but the joints between the proximal and distal phalanges are longer than usual. The thumbs also are somewhat shortened. All the nails appear to be normal. The toes, in superficial



BRACHYDACTYLOUS HANDS AND FEET IN KINDRED A

Figure 22

X-Ray photographs show the brachydactylous hands and feet of K. H., a 20 year old man in kindered A.

appearance, approach normality more than the hands. All the toes have only two phalanges. The patient feels no handicap in his daily life as a result of these malformations.

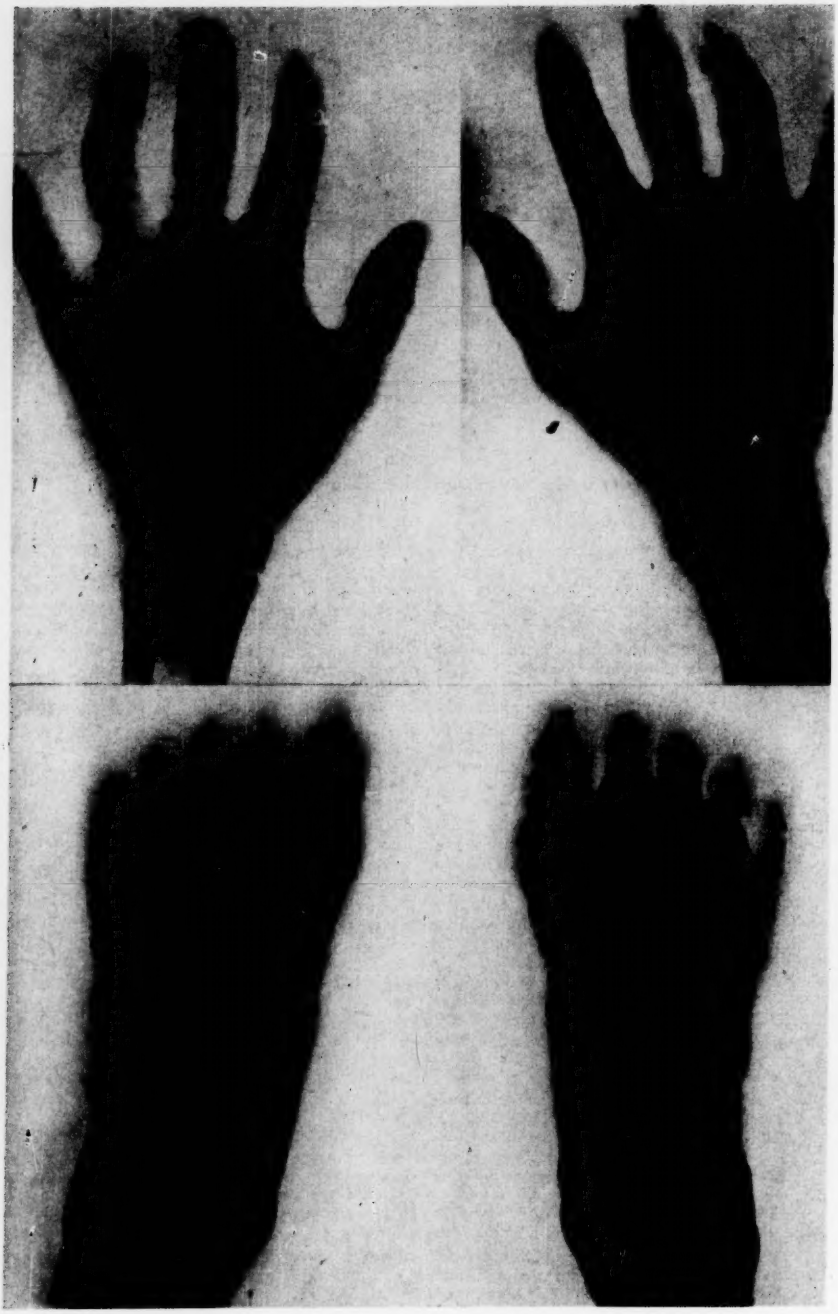
The X-ray photographs (Figure 22) clearly show that the malformation in this man's hands and feet belong to the same category as those reported by Farabee, Drinkwater, Webb and Iltis. The middle phalanges of all fingers and toes are considerably shortened, and devoid of epiphyses. In the hands these phalanges are nearly cubical. More precisely, the middle phalanges of the index and ring fingers are trapezoidal. In the index fingers this bone is slightly shorter on the outside than on the inside, so that the fingers are curved toward the outside; the phalanx of the left hand is ankylosed to the distal phalanx. In the ring fingers the rudimentary middle phalanges are distinctly shorter on the median side than on the outside, so the fingers are definitely curved toward the median side. The middle phalanges or little fingers are completely ankylosed to the distal phalanges, so that these fingers appear like thumbs in X-ray figures. A sesamoid bone is attached to the inner side of the distal end of the metacarpal bone of each thumb. The feet show a distinction from the hands in the complete ankylosis of all middle phalanges to distal phalanges, and all the toes have only two segments like the big toes. A sesamoid bone is found inside the distal end of the first metatarsus on each side.

A girl of eleven, T. H. (VI-17), was interviewed by Mr. Yamazaki. She is healthy, without any physical defect except for her deformed hands and feet. Her stature is shorter than the average of her class by 1 cm., but she surpasses the average in sitting height (by 2 cm.) and body-weight (by 2 kg.). Her fingers, especially the distal parts, are slender. The X-ray photographs (Figure 23) show great similarity to the hands of her cousin (VI-17) described above, except that the bones are more slender and the ossification is still imperfect, and all epiphyses are distinct from the shafts. The middle fingers have longer middle phalanges than any of the other fingers. The middle phalanges of the index and ring fingers are more distinctly trapezoidal than in her cousin's hands, so that the curvature of these fingers is more pronounced. The epiphyses of the distal phalanges of these fingers are still distinct, and discoidal. The middle phalanges have no epiphysis. The little fingers show a discoidal bone inserted between the proximal and distal phalanges. Judging from its thickness, as compared with the thickness of the epiphyses of the distal phalanges, as well as the thickness of the rudimentary middle phalanges in other fingers, this bone apparently represents the middle phalanx. If so, the little fingers are devoid of the epiphyses belonging to the distal phalanges, or the epiphyses are ankylosed to the shafts at a relatively early stage. At any rate, the little fingers differ in this respect from other fingers. A sesamoid bone is present near the distal end of the metacarpal bone of each thumb. Probably they are still cartilages, and the X-ray photograph shows them only faintly.

In the feet the middle phalanges of the second to fifth toes are ankylosed to the distal phalanges, although the boundaries between them remain distinct in the second to fourth toes. In the big toes the epiphyses have not ankylosed to the distal phalanges. There is a distinct sesamoid attached near the distal end of the first metatarsus on the median side.

Kindred B

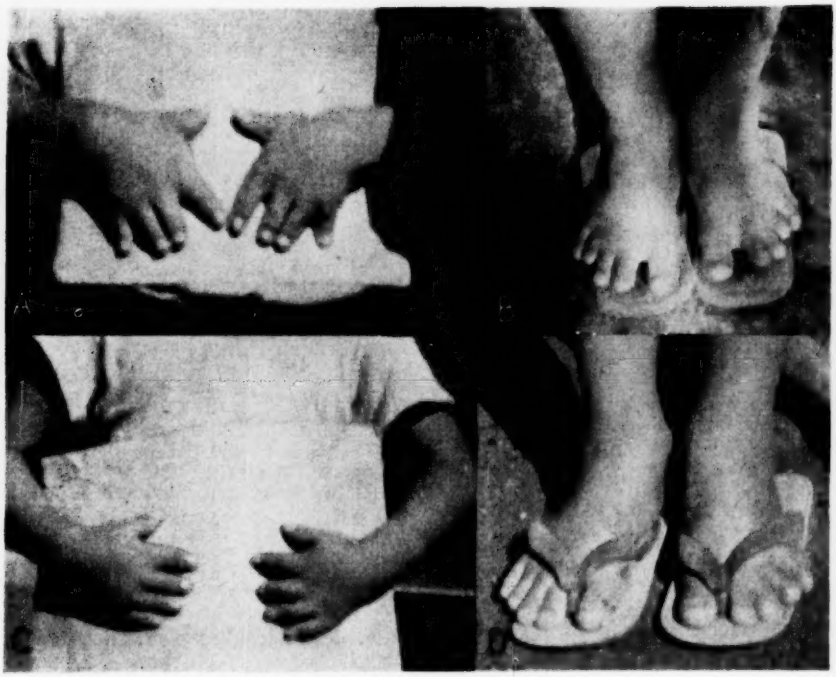
The home of this kindred is at Taura near Yokosuka. The relatives are distributed in the Prefectures of Gumma, Totiki and Saitama.



BRACHYDACTYLOUS HANDS AND FEET OF T. N.

Figure 23

X-Ray photographs show the brachydactlyous hands and feet of T. N., an 11 year old girl in kindred A.



TWO BRACHYDACTYLOUS BOYS

Figure 24

Hands and feet of the two brachydactylous boys in kindred B. *A* and *B*—Y. I., 12 years old; and *C* and *D*—T. I., 9 years old. (Photo by T. Akao).

Since our inquiry could not reach them, this kindred has only been studied partially. This family was discovered by Mr. T. Akao, a student at Nihon University, who published his findings in *Iden* (1950), the popular Japanese journal of genetics. In June of 1951, the writer and Mr. Akao interviewed T. I. and his short-fingered sons.

The oldest member of this kindred to have had this malformation is II-2 (Figure 21 *B*), who was born in 1880 and died in 1945. She married twice, and had by her first husband a son (III-1) who is abnormal. This man is rather thin, about 160 cm. in height and distinctly short-fingered. The woman married a second time, and had two sons and a daugher, besides a son and a daughter who died in infancy. The elder son (III-2) is brachydactylous. He is about 155 cm. in height, his fingers looked much like his sons' to be described below; the toes also resembled his sons', especially those of his eldest son, the fourth and fifth toes being pronouncedly shorter than the other toes. He told the writer that he had not joined the army because of these deformities. The younger sister (III-4) of III-2 was also abnormal; she married her cousin, and has a normal son.

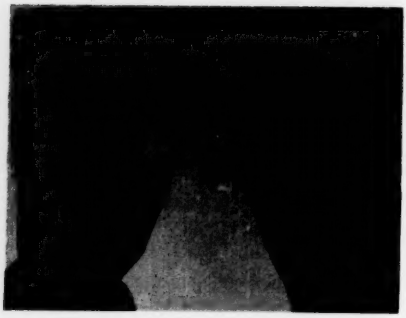
The younger sister (II-6) of II-2 was apparently abnormal; she married a man living in Aomori Prefectures. This couple have three sons, of which the eldest (III-7), a resident of Gumma Prefecture, is reported to be short-fingered. The youngest son (III-10) is the husband of III-4 recorded above.

III-2 married a normal woman, and has three sons to be described below.

Description of the Anamoly of Kindred B

We interviewed all of the sons of III-2, Y. I. (IV-1), T. I. (IV-2) and N. I. (IV-3).

Y. I. is in the first year of a local middle school. He is somewhat below the average of his class in stature, but has a taller sitting height, larger chest circumférence and greater body weight than the average. His hands look plump; the fingers are short and apparently consist of two phalanges. The index fingers are longest of all, and the middle, ring and little fingers are gradually shorter (Figure



ASYMETRICAL BRACHYDACTYLY

Figure 25

The asymmetrical brachydactylous right hand of a 10 year old boy. As far as is known a case similar to this has not been previously recorded. (Photo by N. Nara).

24 *A*). The toes are also shortened and two-segmented. The fourth and fifth toes are particularly small, and they are distinctly receded from the general row of the toes (Figure 24 *B*).

T. I. is in the fourth year of a local primary school. He is much below the average of the class in stature, chest circumference, sitting height and in body weight, standing in stature the third from the last of his class. His fingers are similar in appearance to his elder brother's (Figure 24 *C*). The toes are likewise shortened, and two-segmented; but they are somewhat different from his elder brother's toes in that the fourth and fifth toes are only slightly shorter than the other toes. An X-ray photograph was taken of the hands of this boy, but unfortunately the film was old, and failed to produce a clear picture.

N. I. is a five-year old boy. His hands and feet closely resemble his brothers', especially the eldest brother's, in that the fourth and fifth toes are distinctly smaller than the rest.

Kindred C

The writer owes thanks to Prof. G. Yasuzumi and his associates at Osaka University for all the information concerning this kindred. The family are living in Asiya between Osaka and Kobe. They were extremely unsocial, apparently being overly conscious of their physical defect. Prof. Yasuzumi visited their home, and interviewed two individuals displaying the anomaly, a woman of seventy (III-4) and a man of fifty-four (III-9), and found their hands distinctly brachydactylous. He also heard from a neighbor that the woman had short toes. The pedigree compiled by Dr. Yasuzumi and his associates is reproduced in Figure 21 *C*. It seems certain that the malformation in this kindred also belongs to the category of typical brachydactyly.

Remarks

There is no doubt that the deformities found in the above three kindreds belong to the category of typical brachydactyly, (brachymesophalangy according to Pol's terminology). The descriptions of the malformed hands and feet given above, and the evidence displayed by the X-ray photographs, show this distinctly. In the three kindreds the middle phalanges of all fingers and toes are distinctly shortened, and devoid of epiphyses, and are often ankylosed to the distal phalanges, so that the fingers and toes actually have only two segments. Otherwise the patients are normal, except for the stature being somewhat below average.

As for the mode of inheritance of this abnormality, there is no doubt that it is caused by a dominant autosomal gene of perfect penetrance, as has been reported by previous workers. There has been no case of marriage between affected individuals in any of the families involved in this report.

In Table I are shown the progeny of the matings in the three kindreds, excluding the cases where the progeny are only partially known. In kindred A there are altogether 11 normal and 12 abnormal individuals, and in kindred B, seven normals and seven abnormals. Thus, the proportion of normal and abnormal individuals is very near the 1:1 ratio. Kindred C is the most fragmentary one. In this there is a case of mating between an abnormal man and a normal woman which produced four abnormal and two normal progeny besides three unclassifiable individuals.

Summary

Three Japanese kindreds of typical brachydactyly are recorded. They are residents in rather distant localities, and there is no reason for suspecting any mutual relationship. Their

TABLE I. Numbers of normal and abnormal progeny from the matings of abnormal × normal

Kindred	Number of matings	Progeny: Normal ♀	Normal ♂	Abnormal ♀	Abnormal ♂
A	5	5	6	8	4
B	6	1	6	1	6
C	1	2	0	2	2

abnormality belongs to the typical brachydactyly previously recorded by Farabee, Drinkwater and Iltis. There are some differences in details of the deformity which distinguish kindreds A and B. Kindred A closely resembles the type of deformity reported by Iltis and the first kindred reported by Drinkwater.

All marriages of the affected people are with normal spouses, and have produced nearly equal numbers of normal and abnormal progeny. Kindred A extends over four generations, and contains 11 normals and 12 abnormals produced from such matings; Kindred B extends over three generations and contains seven normals and seven abnormals; Kindred C also extends over three generations, and contains two normals and four abnormals besides three unclassifiable members produced from one such mating.

Addendum

When the writer was nearly through this manuscript, he received the photograph produced in Figure 25 sent by Mr. N. Nara, a student at Hirosaki University. These are the hands of a ten-year old boy in the fourth year class of a primary school in Aomori Prefeature. As shown in Figure 25, this is a case of asymmetrical brachydactyly, affecting the right hand only. All the fingers of this hand are only about three-quarters as long as the corresponding fingers of the left hand. This is primarily due to the degeneration of the middle phalanges, (X-ray photographs not shown here). The degeneration, however, is of a smaller degree than in the cases previously described. The phalanges in this case are nearly twice as long as they are broad and are devoid of epiphyses. The little finger is strongly curved in the part corresponding to the middle phalanx. Only the thumb shows no sign of degeneration. The metacarpal region is slightly shortened.

According to Nara, this boy is the only member in the family having this malformation. He has two brothers and a sister who are all normal, besides another sister who died in infancy. The parents are cousins, and both are normal. The boy's grandparents, uncles and aunts and cousins, so far as could be ascertained, were all normal.

It is possible that the brachydactyly of this boy was caused by some accident in development, and has no hereditary basis. It is noteworthy, however, that it resembles in its appearance those hereditary cases already described in that all the middle phalanges are shortened, and are devoid of epiphyses. It is also striking that only one hand is affected. The writer does not know if such a case has previously been recorded.

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CONCORDANCE OF IDENTICAL TWINS IN SALIVARY ACIDITY-ALKALINITY

Monozygotic Twins and Siblings Compared in Respect to Salivary Tests For Titratable Alkalinity and Titratable Acidity

NAOMI C. TURNER, JACK T. BELL, JAMES H. SCRIBNER AND JACQUES-YVES MEYER*

THE data reported here came to light in connection with a study of saliva in relation to dental caries. In the course of testing the saliva of 315 children aged five to 11 years for titratable acidity and titratable alkalinity, 18 pairs of monozygotic twins were included in the test series. Fortuitously, 18 sibling sets were also part of this study. Two sets of fraternal twins are among the 18 sibling sets.

Methods and Materials

The saliva used for testing was whole saliva as it comes from the mouth. Collection was directly into clean dry test tubes. At least two hours had elapsed after the last meal, or other food, before collection. Since a diurnal variation in pH has been reported by Eisenbrandt[1], collection was either between 10 and 11 A.M. or 2 and 3 P.M. The production of saliva was not stimulated by paraffin chewing. Marshall[2] has reported that saliva so produced varies in titratable acidity and titratable alkalinity from resting saliva. It was whole, resting saliva which we sought to measure for acidity and alkalinity.

Titratable acidity was measured with .001N NaOH, phenolphthalein indicator. Titratable alkalinity was measured with .001N HCl, methyl red indicator. For each test 1 ml of freshly collected saliva was used.

Results

The primary point of interest in this paper is a comparison of the saliva of the monozygotic twins versus a set of siblings who are not monozygotic twins. In order to appreciate the salivary differences it is necessary to consider the duplicability of the test procedure. Two operators worked interchangeably, the reagents were carefully prepared and checked, but here, too, one must allow for a possible, though small, source of error. In addition to the variables of interchangeable operators and reagent batches, we are of course dealing with a functional organism as our source of test material.

In the course of testing, 23 children were sent to us from the admitting room a second time (1, 2, 3, or 4 weeks after the child's first test). Purposive selection was not involved. The results of these retests are shown in Table I. The results of similar tests for the siblings and twin groups are given in Tables II and III.†

We applied a test of variance to these three groups. The formula used in the computations was that of:

$$\text{Variance of difference} = \frac{d^2}{2N}, \text{ where}$$

d = difference of individual measurements
N = number of paired comparisons

Applying the "F" test[3] we find that in titratable acidity the variance of the difference for retests and twins is not significant while the variance of the difference for twins and siblings and for retests and siblings is significant at 1 in 1000. In other words, while retests and twins are very similar, siblings and twins, or siblings and retests are significantly different at .001. In titratable alkalinity the variance of the difference for the twins and retests show an "F" test at .05; siblings and

*Biochemistry Laboratory, Forsyth Dental Infirmary for Children, 140 The Fenway, Boston, Massachusetts.

†Tables I and III are only partially presented herein. Table II is presented in its complete form because of the importance of the twin data. Tables I, II, and III are available as supplementary documents. To obtain this material, order Document No. 3970 from the American Documentation Institute Auxiliary Publications Project, Photoduplication Service, Library of Congress, Washington 25, D. C. Remit $1.25 for microfilm (images one inch high on standard 33 mm. motion picture film), or $1.25 for photoprints (6 × 8 inches, readable without optical aid).

twins show variance of difference of border line significance; while siblings and retests show variance of difference at .001. (See Table IV).

Discussion

A question that one might immediately raise concerns possible sex difference. An inspection of the frequency distribution of the 315 cases[1] does not indicate that we are dealing here with a sex difference. A matter of interest is the extent to which these salivary tests reflect systemic characteristics. Since variance of difference for twins in titratable acidity is less than for siblings, and similar to retests, one may be justified in interpreting the test as a reflection of systemic or physiologic state.

Summary

As part of a study of 315 children aged five to 11 years, titratable acidity and titratable alkalinity of fresh, resting whole saliva were measured for 18 pairs of monozygotic twins, 18 pairs of siblings, and 23 individual children repeated a second time (in one to four weeks).

Analysis of variance for test readings of titratable acidity show twins and retests to be more similar than siblings and retests or siblings and twins. Thus titratable acidity of the saliva would seem to measure not a chance variant but rather reflects an inherited type of systemic activity.

TABLE I. Titratable alkalinity and titratable acidity for 23 individuals (not siblings) whose salivary tests were repeated a second time after an interval of one to four weeks

Name	Age	Sex	Alk.	Ac.	Interval (wks.)
E. B.	8-6	F	13.5	1.55	2
			14.8	1.1	
R. B.	9-1	M	12.3	2.0	2
			11.3	1.1	
W. C.	7-7	M	12.98	2.15	2
			16.6	3.1	
R. C.	7-9	M	14.4	.6	3
			14.7	.9	
F. G.	7-6	M	6.75	2.2	3
			8.9	1.0	
P. G.	10-2	F	5.0	4.0	1
			5.83	3.8	
A. J.	8-8	F	7.9	6.45	4
			7.9	6.7	
E. K.	9-11	M	9.95	2.2	1
			8.6	2.5	
D. K.	10-7	F	9.4	2.6	1
			9.1	3.1	
E. K.	7-7	F	10.1	2.2	2
			11.4	3.1	

TABLE III. Titratable alkalinity and titratable acidity of the saliva for 18 pairs of siblings (part of a study of 315 individuals including 18 sets of monozygotic twins)

Name	Age	Sex	Alk.	Ac.
M. A.	7-11	F	13.4	3.3
S. A.	9-9	M	21.2	2.4
C. M.	6-6	F	7.29	3.0
W. M.	11-3	M	6.75	5.15
F. C.	6-5	F	9.9	1.5
C. C.	7-9	F	7.15	2.4
W. C.	6-4	M	14.1	2.1
J. C.	7-4	F	12.6	4.45
P. D.	5	M	7.0	3.25
M. D.	8-5	F	10.5	2.85
M. G.	6-11	F	11.15	2.2
P. G.	8-8	F	12.6	2.45
P. H.	8-9	F	14.1	2.2
J. H.	6-5	M	11.65	1.05
C. H.	9-2	M	11.65	2.50
P. H.	10-6	F	10.6	3.10
G. N.	8-4	M	7.9	5.15
A. N.	9-2	M	8.1	1.7
M. J.	9-8	F	11.9	3.5
A. J.	8-8	F	7.9	6.45

TABLE II. Titratable alkalinity and titratable acidity of the saliva for 18 sets of monozygotic twins

Name	Age	Sex	Alk.	Ac.
J. B.	5-8	M	8.6	1.42
C. B.	5-8	M	7.7	1.65
R. G.	5-6	M	9.9	2.7
R. G.	5-6	M	8.5	2.6
K. G.	5-1	M	10.1	2.1
K. G.	5-1	M	8.0	2.4
G. K.	5-11	M	12.9	2.7
C. K.	5-11	M	9.9	2.6
W. W.	6-0	M	11.7	3.8
W. W.	6-0	M	11.1	2.7
A. H.	7-4	M	9.4	4.0
A. H.	7-4	M	12.4	3.2
F. M.	7-11	M	8.8	1.8
J. M.	7-11	M	9.1	1.6
E. C.	10-4	M	9.6	3.1
W. C.	10-4	M	10.3	2.8
D. H.	10-10	M	11.7	2.6
D. H.	10-10	M	9.9	2.7
T. W.	10-8	M	10.4	1.3
W. W.	10-8	M	12.9	1.5
M. A.*	5-11	F	6.4	4.0
L. A.*	5-11	F	11.0	3.7
P. C.	7-8	F	11.95	1.6
P. C.	7-8	F	9.0	2.5
E. K.	7-7	F	10.1	2.2
K. K.	7-7	F	11.8	2.1
J. W.	8-10	F	5.4	3.5
C. W.	8-10	F	7.25	5.0
J. D.	8-5	F	11.0	8.0
J. D.	8-5	F	7.0	9.0
M. K.	10-5	F	9.4	4.2
R. K.	10-5	F	12.0	3.8
M. W.	8-5	F	10.6	3.2
M. W.	8-5	F	9.31	1.95
A. M.	10-2	F	7.8	2.2
M. M.	10-2	F	9-1	3.8

*One twin was subject to severe asthmatic attacks, the other was not.

TABLE IV. Variance of the difference between individual mearurements of alkalinity and acidity in siblings, twins and retests

	Alkalinity		Acidity		
	Variance	"F"	Variance	"F"	N
Sibling Comparisons	5.789		1.349		18
MZ Twin Comparisons	2.719		.295		18
Retests on Single Individuals	1.002		.239		23
Sibling/Twin		2.1290[a]		4.572[d]	36
Sibling/Retest		5.7774[d]		5.644[d]	41
Twin/Retest		2.7135[b]		1.234[c]	41

[a]not quite .05
[b]beyond .05
[c]not significant
[d] beyond .001

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(*Received August 11, 1952*)

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HETEROSIS

HETEROSIS* is an attractive and well printed volume that contains thirty papers on subjects bearing more or less closely on the subjects of hybrid vigor. These were presented by as many workers or groups of workers at a conference held at Iowa State College during the June 15-July 20 period of the summer of 1950. Unfortunately it was not feasible to include accounts and discussions of these papers or of the accompanying "methods workshop." Nevertheless Heterosis is a volume that all geneticists will want to have available, whether they are interested in plant and animal breeding or in other aspects of the subject.

Heterosis begins with an important and entertaining account by Zirkle of ideas on hybrid vigor from the Greeks to Mendel. There follows a chapter by Shull recounting the way in which the concept of heterosis developed in his mind and the relation of his work and thinking to that of his contemporaries. Completing a series of three chapters on historical aspects of the subject is a paper by Hayes which emphasizes the development of theories and applications of heterosis after 1910.

The remaining 27 chapters cover various aspects of the subject and show a wide range of quality. Three deal only indirectly with the subject. One by Brink deals with seed development in relation to heterosis. Whaley, Robbins and Emerson contribute three papers on the physiological and biochemical basis of hybrid vigor. Various phases of the genetics of the phenomenon are considered in a series of eight chapters by Smith, Jones, Irwin, Crow, Powers, A. J. Mangelsdorf, Gowen, and Comstock and Robinson. The origin and development of corn in relation to heterosis is considered in chapters by Anderson and Brown, P. C. Mangelsdorf and Wellhausen. General genetic and population aspects of the problem are treated by Buzzati-Traverso and by Dobzhansky. Finally seven chapters by Dickerson, Henderson, Winters, Pinnel *et al.*, Chase, Sprague and Hull are devoted primarily to problems involving applications of heterosis in plant and animal breeding.

For more than a third of a century there have been two hypotheses as to the genetic basis of heterosis. One assumes that complementary dominant genes favorable for growth, and acting additively or synergistically, account for the phenomenon. The other postulates that heterozygosity *per se* is somehow responsible. The present trend is to believe on the basis of various lines of evidence that heterosis results from a combination of both types of gene action. There are many instances known of complementary gene action influencing growth. In a number of these, for example in Neurospora, the interaction can be described in terms of the ability to synthesize specific metabolites. In the volume under review a biochemical model for one-gene heterosis is described by Emerson. In this a Neurospora heterocaryon (physiological approximation to a heterozygote in a diploid organism) with a one-gene difference between the component nuclei shows superior growth to either homocaryotic parental strain. Biochemically it appears that one homocaryotic strain synthesizes too little of the vitimin para-amino benzoic acid, whereas the other produces an excess that is used to promote a reaction deleterious to growth. The heterocaryon achieves a proper balance in which sufficient vitamin is made for growth but not enough for the deleterious reaction. It is possible that this kind of situation in which overproduction of a specific substance leads to a decrease in growth may be of considerable general importance in heterosis.

The term "overdominance" is often used for situations in which heterozygosity for a specific gene is more favorable for growth than is either homozygous condition of the same gene. Emerson's model makes clear the danger of using a term that implies a specific interpretation in situations in which there is no basis for such an interpretation. With respect to growth in the Emerson case one would say that the normal allele of one of the genes concerned with synthesis of para-amino benzoic acid is "over-dominant." But there is obviously no overdominance with regard to vitamin synthesis, a process without much doubt closer to the primary action of the gene than is total growth response. On the contrary, incomplete dominance is clearly indicated. It seems to the reviewer that this example shows that the term "overdominance" as used currently is an unfortunate one.

G. W. BEADLE
California Institute of Technology

**Heterosis.* John W. Gowen, Editor. Iowa State College Press, Ames, Iowa, 1952. $5.75.

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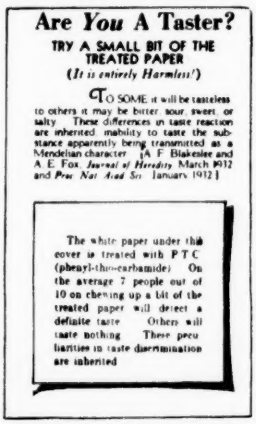
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